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SOME REMARKS ABOUT DIZZINESS – ITS DIAGNOSTIC SIGNIFICANCE AND TREATMENT

W. J. McNALLY, M.D.

MONTREAL, CANADA

When invited by your Committee to address you, it was intimated that I should speak on the subject of dizziness or vertigo. I agreed, with mental reservations, because of the difficulty I have in developing or clarifying my ideas about this involved condition.

Williams[39] pointed out that dizziness derives from the old English word "dizzy" meaning foolish or stupid, whereas vertigo derives from the Latin word "vertere" meaning to turn. Most writers, however use the words dizziness or vertigo interchangeably. Adler[1] has stated that dizziness results from conflicting information from the sense organs ordinarily concerned with balance. As the result of the conflict there is confusion at the conscious level, and the confusion results in dizziness or vertigo. Dizziness has also been defined as a consciousness of discord in the postural mechanisms. It can be caused by changes in any of the three systems responsible for balance or posture, namely the ocular, kinesthetic or vestibular mechanisms. One would naturally expect a considerable variety of effects from such a multiplicity of possible sources.

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Attempts have been made to attach diagnostic significance to specific kinds of dizziness. It is usually agreed that a patient who develops a sudden attack of rotatory dizziness, associated with head noises and deafness, is most likely suffering from irritation of the vestibular mechanism. On the other hand, it is a fact that stimulation of the vestibular mechanism, as by the caloric test, does not always cause rotatory dizziness. The patient may describe a feeling of unsteadiness, or he may be more disturbed by the associated vagal stimulation and describe a feeling of weakness or a feeling of confusion.

Adler[1] has stated that ocular dizziness is never severe. It is frequently of long duration and is usually associated with other signs of ocular disease. Dizziness arising from a disturbance in the kinesthetic mechanism may also be associated with other signs of disturbance in that system. Dizziness, which arises from disturbance in the central nervous system may be difficult to differentiate from an attack of peripheral labyrinthine dizziness. The latter occurs in short attacks and frequently is associated with nausea and vomiting. The association of dizziness with head noise, and, or hearing loss is a good indication that the dizziness is of peripheral or labyrinthine origin—a better and surer indication than is the patient's description of its character.

The diagnosis is made more difficult by the fact that one may have involvement for a long time of either the cochlea or the labyrinth before the other system is involved. This possibility makes it necessary to be continually on the look-out for additional symptoms which may clarify the diagnosis. At the present time, the diagnosis of Ménière's syndrome or labyrinthine hydrops depends more upon the results of the hearing tests than upon the vestibular tests. This has resulted from the work of Dix, and Hallpike,[8] and will be referred to later in connection with the recruitment test and speech audiometry.

Because there is still some confusion about the parts played by the different labyrinthine end organs in producing dizziness and nystagmus, and their associated signs and symptoms, a brief review of the generally accepted ideas of the physiology of the semicircular canals and of the otoliths is in order.

Semicircular canals. The three semicircular canals which are situated in each ear, at right angles to each other are normally stimulated by acceleration—the rate of change of velocity. Steady turning does not stimulate a semicircular canal. It is the change in speed

of turning above a certain threshold which acts as a stimulus. The rate of change can be kept so low that a canal is not stimulated. If the subject is placed over the axis of rotation, one may apply a subthreshold acceleration, and the velocity may become high, without stimulating the semicircular canals. If, however, in an experiment similar to the above the subject being examined is placed in a position away from the centre of rotation, a second stimulation, viz. centrifugal force, is brought into play. This, together with gravity, which is constantly acting, does exert a stimulus upon the labyrinth. It does not stimulate the semicircular canals, but it does stimulate the utricles—Tait and McNally.[37] This factor will be discussed later in connection with the utricle.

The semicircular canals respond to acceleration, which is their normal physiological stimulus, but they are also stimulated by change of temperature, which is the stimulus in the caloric test. They are stimulated by mechanical pressure or decompression. This is the effective stimulus in the fistula test. They respond to electrical stimulation as in the galvanic test. The labyrinth is stimulated by any interference with its blood supply or by toxic or irritating substances which may reach it directly or by means of its blood supply.

Ewald[12] found that the semicircular canals of the pigeon are stimulated by turning in either direction, but that a turn in one direction is more effective than a turn in the opposite direction. His work may be summarized by stating that a movement of the endolymph towards the ampulla in a horizontal canal is a more effective stimulus and elicits a greater response than does movement of the endolymph away from the ampulla. On the other hand, movement of endolymph in a vertical canal away from the ampulla is a more effective stimulus than a movement of the endolymph towards the ampulla. The more effective stimulus in a canal causes a quick phase nystagmus of the eyes towards the homolateral side, and the nystagmus is in the plane of the stimulated canal, or in a resultant plane if two canals in different planes are stimulated simultaneously. Flourens had previously noted that stimulation of a semicircular canal tended to elicit a reaction in the plane of the stimulated canal. The findings of Flourens and Ewald have been generally accepted as the basis for interpretation of the clinical tests of the human labyrinth. Experimental investigation carried out upon the labyrinth has shown that the nystagmus which follows from vestibular irritation is the result of semicircular canal stimulation. The better controlled experiments carried out upon the otoliths, the saccule and the utricle, have not resulted in nystagmus. This matter has been discussed in detail by McNally and Stuart.[28]

The three semicircular canals in each ear are so arranged that there are two vertical canals and one horizontal canal. For each canal there is an oppositely directed canal in the same plane in the other ear.

McNally and Tait[30] showed that when one of the frog's semicircular canals is stimulated, after all the other canals have been eliminated by nerve section, the single unopposed canal brings about a very definite overcompensation. The canals are connected to the protective musculature on both sides of the body. Normally, the stimulation of the opposite canal tends to counter-act that over-protective action, and there results the exact amount of reaction necessary for proper body adjustment.

The Saccule. The saccular otoliths which lie in a vertical plane, have not been shown to have a vestibular function. Some well controlled experiments in animals would suggest that the saccule may be stimulated by vibration. With our present knowledge it can be excluded from a discussion of the vestibular mechanism—McNally and Stuart.[28]

The Utricle. The utricular otoliths, which lie in a horizontal plane, are responsible for the so-called static or gravity reactions. The actual mechanism of stimulation of the utricle is not known. Tait and McNally[37] showed that it is not stimulated by a turning movement in the horizontal plane about a vertical axis if the animal is over the centre of rotation. On the other hand, the utricle does react during the turning if the animal is placed away from centre, so that centrifugal force is acting. To show this, a frog, in which the nerves to all six semicircular canals had been cut, but with the utricles intact, was placed in an eccentric position on a turntable. As the speed increased the animal leaned in towards the centre of rotation and carried out the protective reactions of a normal animal in response to the increasing centrifugal force. If a frog, with denervated canals, but with intact utricles, is tilted slowly out of the horizontal plane it will react to the force of gravity by re-adjusting its legs in such a way as to maintain its head level. On the other hand, if such a frog, without semicircular canals, is given a sudden tilt out of the level position it will throw itself in the direction of the tilt, making an anticompensatory reaction in response to angular acceleration. Apparently this reaction is designed to tone down the over-compensation of a single unopposed semicircular canal. A frog with only utricles intact, and in which the nerves to all six semicircular canals have been severed responds normally when subjected to linear acceleration, either in the horizontal or vertical plane. If

the nerves to the utricle on each side are severed, a frog can no longer respond normally to slow tilting about a horizontal axis or to centrifugal force, or to linear acceleration.

Versteegh[38] established that in the rabbit, severing the nerves to the utricles eliminated all the static or positional reflexes which had previously been ascribed to both the saccule and the utricle. These reactions are the counter-rolling compensatory movements of the eyes in response to change of position of the head, the labyrinthine head-righting reflexes and the body musculature readjustments in response to slow changes of position of the head.

DeKleyn and Lund[6] carried out simultaneous bilateral caloric stimulation of the semicircular canals of the rabbit. At the same time they found that the animals still showed normal reactions of the eyes and limbs to tilting movements, which should stimulate the utricles. In a second set of experiments, after centrifuging away the otolithic membranes in the guinea pig, they could still elicit normal nystagmoid eye movements during simultaneous bilateral semicircular canal stimulation with the caloric test. They concluded that utricular reactions could still be elicited, even while the semicircular canals were reacting to caloric stimulation and that the caloric and post-caloric nystagmoid reactions could be elicited in the absence of the utricles.

Grahe[19] described two tests to be carried out on a specially constructed tilt table. In one case the patient is blindfolded and strapped to a carriage with the head fixed. He is then tilted into or out of the vertical plane. This test is called "Sensation of the Vertical" and it requires the presence of intact utricles. The second test is to tilt the blindfolded individual, with the head free, out of the vertical plane while the head is being watched for head-righting reactions.

Subjecting a person to centrifugal force at sub-liminal acceleration should stimulate only his utricles and should elicit only utricular symptoms. This was accomplished by Graybiel, Noven and Walsh.[20] They made use of a centrifuge large enough for patients. They could stimulate the semicircular canals by rotation and acceleration about a vertical axis. On the other hand, if they carried out the turning in such a way that the acceleration was maintained below the threshold of canal stimulation, and if the subject's head was properly placed, they could stimulate the utricles by centrifugal force, without stimulating the canals. In addition to testing normal individuals they reported the results of examining five patients who had been diagnosed as having Ménière's disease, and in each of

whom a unilateral labyrinthectomy had been done. With the turning tests they could reproduce the patients' previous spontaneous symptoms. Otolithic symptoms predominated in one of the five cases and were present in two of the others. What is most important is their description of the symptoms which occurred during utricular stimulation and how these differed from those of semicircular canal stimulation. They stated that nystagmus was never referable to stimulation of the otoliths. Stimulation of the utricle caused an illusion of tilting of the body and an apparent displacement of objects, so that they appeared to assume a new position in space.

Stimulation of the semicircular canals elicited a feeling of rotation and an apparent rotation of objects in the visual field. Nystagmus was referable to stimulation of the semicircular canals.

If such tests are well controlled the centrifuge may give valuable information about human utricular function and symptomatology.

Intracranial Connections. The vestibular and cochlear nerves, together form the eight nerve and are in close association until they separate to join their respective nuclei in the brain stem. A small lesion central to the nuclei is unlikely to involve both cochlear and vestibular divisions.

There is a lack of detailed knowledge about the intracranial connections from the labyrinths. Some of the confusion in diagnosis stems from this lack of knowledge. Something is known about the vestibular nuclei, and about their connections with the external ocular nuclei. There is a free connection with the cerebellum. There is an elaborate connection with the body musculature. The connections with the autonomic nervous system are manifested by the pallor, perspiration, nausea, vomiting etc. which accompany vestibular irritation or stimulation. The paths connecting the vestibular nuclei to a possible labyrinthine centre in the temporal lobe are not known in great detail. The medulla and pons are the parts of the central nervous system which are essential for the normal elicitation of most of the known vestibular reflexes.

HISTORY

When consulted by a patient who complains of dizziness, the general history and the story of the present illness must be elicited, together with a clear description of the dizziness. Other symptoms and signs, especially tinnitus and deafness, should be recorded.

Symptoms such as headache, fainting, numbness and diplopia, should be inquired about, and, if present, should be very carefully

assessed. These are not symptoms which result from disease of the vestibular mechanisms, but frequently indicate central nervous system disease. Furstenberg (1952)[15] stressed the importance of properly assessing such associated symptoms. He specified particularly the complaint of numbness of the face and ear on the affected side, which points to involvement of the fifth nerve. He also mentioned a burning sensation in the throat, which could refer to an associated involvement of the glosso-pharyngeal nerve or its connections.

McNally, Stuart, McKercher, and Lockhart[20] reported in their series of two hundred cases of vertigo that in every case in which numbness or loss of consciousness was present, in association with dizziness, there proved to be organic disease of the central nervous system.

Some important points to be covered in the history are—suppurative ear disease, upper respiratory infection, allergy—personal and family, serious systemic illnesses, accidents, operations, drug intake and medication. The patient's habits and occupational hazards should be inquired about, especially regarding the use of tobacco and alcohol. The family history of ear trouble, and the possibilities of congenital defects must not be overlooked.

EXAMINATION

In the general physical examination particular attention should be given to the cardio-vascular and central nervous systems. The importance of the general examination cannot be over-emphasized. In our[29] review of two hundred cases of vertigo evidence of serious cardio-vascular disease was found in about twenty-eight percent, organic disease of the central nervous system was found in twelve percent, and psychosomatic disease in about ten percent. The examination of the ears, nose, throat and larynx should include x-ray studies of the mastoids, and of the base of the skull to show the internal auditory meatuses.

Vestibular Tests. The eyes should be examined for spontaneous nystagmus in various positions. The presence or absence of positional nystagmus is particularly important if the patient has given a history of postural dizziness—that is if the dizziness is associated with any special head position.

Tests for incoordination, adiadokokinesia, ataxia, inequality of pupils, corneal reflexes, etc. should be included at this time.

In order that the vestibular tests will not be put aside to another day, one should become familiar with a simple test. If the test

allows ease of repetition, the irritability of the labyrinths can be determined from time to time. The simplest vestibular test is some form of the Kobrak minimal stimulation, either with cold water or with cold air. If one uses a test routinely, a standard can be established by which can be judged hypo and hyper irritability of the labyrinth. If something develops in the test which is not clear, more elaborate tests can be carried out.

Dix, and Hallpike[8] reported that the caloric tests (alternate hot and cold stimulation by the technique described by Fitzgerald and Hallpike,[13] showed some abnormality in ninety-four percent of their cases of Ménière's syndrome. Other authors do not report such a high proportion of abnormal results. Williams[39] pointed out that the labyrinthine tests have not yet reached a high degree of accuracy. There are many physical factors outside the internal ear which will cause slight changes in the duration of the nystagmus. Only marked degrees of difference are significant and they will be indicated by most of the minimal cold caloric tests.

Hearing Tests. The hearing tests have taken on greater significance—Dix, Hallpike, and Hood.[10] In addition to pure tone audiometry, speech audiometry and tests for recruitment may be of diagnostic significance in differentiating between a case of true Ménière's syndrome, on the one hand, and a possible eighth nerve tumor, on the other. It is frequently stated in the literature that there is not much chance of confusing the dizziness which occurs in an eighth nerve tumor with the dizziness of true Ménière's syndrome. In most instances, dizziness of the rotatory type is not a pronounced symptom in eighth nerve tumor. On the other hand, McNally and Stuart[27] reviewed the case reports of thirty-one cases of eighth nerve tumor. There was a history of dizziness in seventeen, and in six of these there was a history of varying degrees of sudden rotatory dizziness. In one patient there was a history of sudden attacks of rotatory dizziness, deafness and tinnitus for two years before any signs diagnostic of an eighth nerve tumor developed. Eventually this patient was operated upon for an eighth nerve tumor.

OTHER SYMPTOMS AND SIGNS

Tinnitus. Tinnitus may precede or follow the onset of dizziness. Sometimes the tinnitus is a more disturbing symptom to the patient than is the dizziness. Medical treatment rarely brings about any relief of the tinnitus. Sometimes patients will report that the tinnitus has improved, but the improvement may bear no relation to the treatment.

Labyrinthine ablation or labyrinthectomy is not followed by relief of tinnitus in any more than fifty percent of cases. Dandy[4] reported relief of tinnitus in fifty percent of the cases in his series of nerve section, and it should be noted that in some cases where both eighth nerves were sectioned the tinnitus persisted. In the follow-up of Dandy's series, reported by Green and Douglas[21] the tinnitus ceased in twenty-seven percent of the cases submitted to partial nerve section, and in thirty-seven percent submitted to complete nerve section. It is this persistence of tinnitus following a treatment which may improve the dizziness, which suggests that the pathological changes of Ménière's syndrome may not be confined to the peripheral labyrinth.

Deafness. The hearing loss associated with dizziness and which is characteristic of Ménière's syndrome is usually a perceptive type of deafness. It may be a low or a high pure tone loss and it is accompanied by a loss of bone conduction. In spite of the perceptive character of the loss a predominant feature is the tendency to fluctuation of hearing from time to time. This has been emphasized by many observers.

With the advent of speech audiometry certain characteristics became associated with perceptive deafness. In addition to an elevated threshold for conversation, there may be a lowering of discrimination or intelligibility at higher intensities, and a lowered threshold of discomfort. In other words, the perceptively deafened individual is not necessarily helped by increasing the loudness of the conversation above a certain point, and may actually be made uncomfortable by the loudness.

Recruitment. Dix, Hallpike and Hood[9, 10] and Dix, and Hallpike[8] showed that the above remarks are particularly true in cases of Ménière's disease. Furthermore, they showed that whereas the binaural loudness recruitment test of Fowler[14] which had previously been considered diagnostic of perceptive deafness generally, applied particularly to cases of Ménière's syndrome which they regarded as a labyrinthine end organ disease—a disease of the hair cells in Corti's organ. Many cases showed over recruitment—the sound being heard louder in the diseased ear. On the other hand, they reported that other cases of perceptive deafness, such as in cases of eighth nerve tumors, which they class as nerve fibre disease, do not show recruitment. These findings have been confirmed by Eby and Williams[11] Reger and Kos[35] and others. With the report of the above work, there has been a great revival of interest in the recruitment phenomenon. Recruitment refers to the phenomenon in which the compar-

ative loudness, in the two ears, of a given tone which at threshold was unequal, tends to become equal with increased intensities. The poorer ear is said to recruit. Many other methods have been developed to estimate the phenomenon of recruitment. There is also a monaural loudness balance in which two tones of different frequencies are compared in the same ear at varying intensities. This test was described by Reger.[31] More recently there have been a number of tests advocated, the most prominent among which is the difference limen test usually associated with the name of Bekesy. This last test deals with the determination of variations in the estimation of threshold intensity difference. Reger and Kos (1952[35] have described Bekesy's audiometer as "a self-testing, automatic recording, continuously variable frequency audiometer, which records graphically the intensity range over which the test tones must be increased and decreased to be perceived and not perceived. The advantages of this test are that it is applicable in any type of unilateral or bilateral hearing impairment, and the record, in addition to recording the intensity loss over the range of hearing tested at the same time records the difference limen, which is also an index of the amount of recruitment present. In the presence of recruitment the loudness is increased more quickly with increasing tone intensity, so that a relatively small increase in intensity is required for the tone to be perceived. In this way the variations in intensity recorded on the audiogram are smaller than if the recruitment phenomenon were absent. These variations in intensity serve as an indicator of the recruitment phenomenon." The various methods will not be considered any further here because many are still in the experimental stages and there is no uniform agreement as to the interpretation of detail and as to the relative value of the various methods.

Diplacusis. Diplacusis, which is the hearing of a given tone differently in the two ears, is regarded by Shambaugh, Jr.[36] as an indication of a lesion in the organ of Corti. He pointed out that the tone is usually heard higher in the affected ear, and there is an alteration in the quality of the tone. It is unpleasant or raspy in the diseased ear. The test is very simply carried out by asking the patient to compare the pitch of a tuning fork held alternately to each ear. Diplacusis is commonly found in Ménière's syndrome.

Nystagmus. Spontaneous nystagmus may arise from ocular and from central nervous system disease as well as from disease of the labyrinth. When it does arise from the latter it is usually of short duration and is often associated with signs of cochlear dysfunction. A non-labyrinthine nystagmus may be of long duration and is usually associated with localizing signs of central or ocular disease.

If an induced labyrinthine nystagmus is abnormal, any change in the amount of reaction may be due to peripheral or end-organ disease, e.g. a hypo, a hyper, or an absent reaction. However, if the character of the reaction is affected the cause is probably within the central nervous system, i.e. if there is any asymmetry in the movement of the two eyes, or if the direction of the movement is not what is expected, etc.

Positional nystagmus and postural dizziness are closely related. As yet their etiology and significance have not been definitely established. Dix and Hallpike[8] stated that Bárány first described positional nystagmus in 1921. Nylén[32] reviewed the literature and pointed out that positional nystagmus occurs in a wide variety of conditions. He found it common in Ménière's syndrome, also following drug intoxication, in cranial trauma, in encephalitis and in brain tumor. He pointed out that the patient must be examined with the head in different positions—head erect, head back, head forward, head on each side and with the head hanging back over the end of the table.

The behavior of the nystagmus in relation to position and movement of the head determined the following classification of Nylén:

Direction Changing	(Type I)
Direction Fixed	(Type II)
Irregular	(Type III)

In the cases of brain tumor which he reported he found that nystagmus of fixed direction (Type II) amounted to fifty percent and nystagmus of changing direction (Type I) amounted to thirty percent. Out of twenty-nine of his cases of Ménière's syndrome, fifteen showed the nystagmus to be fixed and six showed the nystagmus to be changing.

Lindsay[25] stated that Nylén's Direction Changing Nystagmus (Type I) was found chiefly in lesions of the central nervous system, whereas the Direction Fixed Nystagmus (Type II) may occur in peripheral disease or in central diseases. In contra-distinction to Nylén, Lindsay stated that positional nystagmus was rarely seen in true Ménière's disease, and he stated further that positional nystagmus was a relatively infrequent occurrence in lesions known to be situated in the peripheral ear. When it did occur it was Type II—Direction Fixed.

Dix and Hallpike[8] did not describe positional nystagmus in Ménière's disease. They ascribed its origin to utricular disease, and

set up a special classification of dizziness, which will be discussed later.

Lindsay[25] found positional nystagmus to be an almost constant feature in pseudo-Ménière's disease, i.e. vertigo without auditory or neurological signs, probably indicating that these cases are of central origin.

Gerlings[17] considered the problem of positional nystagmus, and reviewed the literature to show that there is evidence to show that both peripheral and central disease may be accompanied by positional nystagmus. He adopted Nylén's classification already mentioned. In cases of middle ear suppuration associated with labyrinthitis, which he reported, he noted the presence of Nylén's Direction Changing (Type I) nystagmus. This is the variety usually associated with central disease.

Because the phenomenon is associated with a certain position of the head or change of position, the otoliths have been assigned a role in the etiology. Before attempting to conclude that positional nystagmus is the result of utricular disease, additional information must be forthcoming. One difficulty is that good clinical tests for utricular function are not in common use.

Directional Preponderance. Directional preponderance is a readiness for nystagmus to occur to one side more than to the other, and for this reason it was called "nystagmusbereitschaft." DeKleyn and Verseegh[7] reported its presence in patients suffering from lesions of one cerebral hemisphere. They demonstrated it by using alternate hot and cold caloric stimulation of the labyrinths. For instance, in a case with preponderance to the right, hot water in the right ear and cold water in the left elicit greater reactions than do hot water in the left ear and cold in the right ear. Fitzgerald, and Hallpike[13] described a similar finding under the name of directional preponderance, and localized the lesion to the temporal lobe. Cawthorne, Fitzgerald, and Hallpike[3] reported finding directional preponderance in twenty-one percent of their cases of Ménière's syndrome. They ascribed the origin to a lesion of the utricle on the side towards which the preponderant nystagmus was directed. Jongkees[24] reviewed the subject and reported finding a directional preponderance in seventeen percent of normal individuals. This last report puts into question its diagnostic significance. If it is utricular in origin it should have been a fairly constant finding in the hundred cases of positional nystagmus, to be discussed later, which Dix and Hallpike[8] ascribed to utricular disease.

Past Pointing. Most vestibular tests are designed to stimulate the semicircular canals, and the reaction which is most looked for is eye nystagmus. Rarely does one examine for reflex reactions in the body musculature. The past pointing test depends upon such a reflex, and it is simple to elicit. It depends upon a compensatory movement of the arms in response to labyrinthine stimulation. Since this reflex passes over an entirely different pathway from the vestibular nuclei than do the eye reflexes, the past pointing gives additional information which may be significant, especially if a central nervous system lesion is suspected. Past pointing has been, at times, erroneously described as a subjective reaction to vestibular stimulation, but, actually it is, as already stated, a compensatory arm reaction. For details of this test the reader is referred to The Manual on Examination of the Labyrinth by McNally and Stuart, page 21.[28]

DIAGNOSIS

Dizziness associated with suppurative middle ear disease should be recognized early because of the potentially serious implications.

It is important to separate out cases of dizziness associated with general organic disease. Dizziness is a frequent accompaniment of cardiovascular disease, the anaemias, central nervous system disease, diabetes, thyroid deficiency, post-traumatic head injury, streptomycin toxicity, etc. Such organic disease does not entirely exclude the possibiliy of a diagnosis of Ménière's syndrome, but it overshadows it in importance. If the organic disease is not detected, treatment of the dizziness will probably be ineffective.

In a large group of consecutive cases of dizziness, according to the review of McNally, Stuart, McKercher and Lockhart[29] serious organic disease will probably be found in about thirty-five percent. The diagnosis of true Ménière's syndrome will be most likely in about thirty percent. The remaining cases—thirty-five percent—are usually grouped under such a heading as atypical or pseudo-Ménière's syndrome. Some writers are very intolerant of this latter grouping. The cases are so classified because they have dizziness in common, without signs of cochlear involvement, or because they probably have other features which indicate they do not belong in the first or second groups. It is also true that some patients, at first classified in this last group, eventually develop signs or symptoms which establish a diagnosis that places them in one of the other groups. The cases in this group, therefore, should be continually under review.

Ménière's Syndrome. Cases of peripheral labyrinthine irritation are grouped under the heading of Ménière's syndrome (thirty percent of cases of dizziness). Usually the patient complains of recurring attacks of dizziness. Frequently the attacks are of sudden onset and the dizziness may be rotatory in character, associated with nausea and vomiting. There is usually an associated tinnitus and a unilateral or bilateral loss of hearing, the characteristics of which have already been discussed. There is usually diplacusis and recruitment. There may or may not be changes in the vestibular tests.

The pathology in such a case that died shortly after an operation for eighth nerve section was first reported by Hallpike and Cairns.[22] They described the picture of distention of the ductus cochlearis, which has come to be regarded as typical of labyrinthine hydrops. Hallpike and his co-workers[8] have since pointed out that in addition to the dilatation of the cochlear duct, there is evidence of damage to the hair cells in the organ of Corti. Lindsay[26] reported associated dilatation of the walls of the utricle and the saccule in a few cases, with occasional herniation of their membranes into the ampullae of the semicircular canals. So far no one has advanced any generally accepted explanation for the occurrence of this picture of increased intra-labyrinthine pressure.

Mygind and Dederding[31] first suggested fluid imbalance as a possible cause. Furstenberg, Lashmet, and Lathrop[16] indicated that the cause was a sodium imbalance. Allergy has been frequently suggested as a possible disturbing cause. Williams[39] considered that the disturbance of physical allergy is probably the most likely cause —a cholinergic preponderance.

Hilger[23] advanced a detailed theory of autonomic dysfunction and spasm of the various labyrinthine end arteries by which he explained some of the variation in symptomatology. Experimental proof for the details of autonomic nerve supply to the labyrinth is still lacking.

Atypical Ménière's Syndrome. When one excludes the cases of dizziness associated with organic disease and the cases of true Ménière's syndrome, one is left with a number of difficult diagnostic problems, (thirty-five percent of cases of dizziness), mostly cases of dizziness without associated symptoms or signs of cochlear disease.

Lindsay[25] grouped these cases under the heading of pseudo-Ménière's disease and suggested that they fall into two sub-classes. About half of his cases were found to have low blood pressure and evidence of vasomotor insufficiency, and were considered as a hypo-

tensive group. The other half showed evidence of hypertension and arteriosclerosis, or intoxication from drugs or infection, while others were of more indefinite etiology.

It was in these cases that he found positional nystagmus (Type I) a constant feature, suggesting that they are of central origin—probably the result of cerebral vascular disturbance.

Dix and Hallpike[8] divided these atypical cases into two subgroups viz. vestibular neuronitis and positional nystagmus.

Vestibular Neuronitis. Under this heading, Dix, and Hallpike grouped cases in which the vertigo was usually paroxysmal, the cochlear symptoms were absent, and the cochlear tests were normal. The vestibular function was always abnormal, including some cases with directional preponderance. The galvanic response was reduced in many of these cases. However, they pointed out that the galvanic response may also be reduced in long standing cases of Ménière's syndrome. There was usually an associated febrile illness or foci of infection. Because of the abnormal response to the galvanic test and for other reasons, they stated that the lesion was probably central to Scarpa's ganglion. The condition was essentially benign and responded well to treatment of focal infection. There was generally recovery in a few years with reestablishment of the caloric tests.

Directional preponderance in the cases of vestibular neuronitis cannot be due to peripheral utricular disease if the lesion is central to Scarpa's ganglion. It is interesting to speculate why a lesion central to Scarpa's ganglion should cause what would appear to be a higher rate of abnormal responses to the vestibular tests than do peripheral labyrinthine lesions.

Positional Nystagmus. This second subdivision of the atypical cases of dizziness, described by Dix and Hallpike[8] included cases which manifested positional nystagmus, but only those cases which corresponded to Nylén's Direction Fixed (Type II) positional nystagmus. They excluded from consideration cases of Direction Changing (Type I) positional nystagmus. Two main characteristics were present in their cases, namely; (1) The character of the nystagmus was essentially paroxysmal. (2) The course of the disease was essentially benign; the patients tended to recover with time and simple sedative measures; there was seldom, if ever, associated intra-cranial disease.

They state that Bárány attributed this condition, not to head movement, but, to head position in space resulting probably from a

disorder of the otoliths. Giddiness occurred on lying down in bed or on taking up a certain head position. There was adaptation, i.e. the symptoms were relieved if the subject remained in the uncomfortable position. Cochlear symptoms were generally absent and the vestibular functions were usually normal.

In their method of eliciting positional nystagmus the patient was briskly placed into the critical position. If it is the position that is so important, it should make little difference how the position is assumed. Usually a latent period preceded the nystagmus and there was usually discomfort and flushing. The nystagmus was rotatory and horizontal towards the under ear. There was a reversal of direction if the patient sat up. The symptoms usually decreased with repetition of the test. In cases with unilateral ear disease the positional nystagmus was directed to the diseased ear. About half of the cases reported by Dix and Hallpike showed ear disease. There were no neurological lesions. There was evidence of infection in some cases. They stated that the pathology consisted of an essentially benign and self-limiting lesion in the vestibular system. They referred to deKleyn's vascular abnormality theory resulting from neck pressure influences etc. To eliminate neck reflexes they used a special swing to get the benefit of different head positions without turning the neck or head in relation to the body. In their opinion this could not be a disease of the neurones because of the normal caloric reactions. They reasoned that since the course was benign a neoplastic lesion was impossible and that it was probably an irritative lesion. Labyrinthine hydrops could be distiguished by the difference in character of the dizziness. The lesion affected the otoliths, hence the caloric responses were normal. From a series of one hundred cases they reported the case of a patient who had suffered from vertigo and internal ear deafness for twenty years. Positional nystagmus to the right occurred when the right ear was undermost. The dizziness which resulted from this position was similar to that of the spontaneous sensations. The vestibular reactions were bilaterally normal. The patient died with a glioma involving the basal ganglia and the upper part of the brain stem on the left side. Histological examination of the right internal ear showed degeneration of the spiral ganglia in the cochlea. The following changes were noted in the maculae of the right utricle and saccule: absence of the otolithic membranes, disorganization of the sensory cells and thickening of the sub-epithelial connective tissues with irregular cellular infiltration.

If this were a self-limited benign disease one might ask whether a highly specialized organ like the utricle could recover from such

an extreme histological change. Cawthorne, Fitzgerald and Hallpike[3] explained the presence of directional preponderance in Ménière's disease as the result of a utricular disorder. They argued that the slow phase of nystagmus, which is a tonic phenomenon, arises partly from utricular stimulation. If that be so, these cases of positional nystagmus due to utricular disease should have shown some definite abnormality of nystagmoid reactions to the caloric tests and there should have been directional preponderance in some cases. Furthermore, if directional preponderance is the result of utricular disease and is present in twenty percent of Cawthorne, Fitzgerald and Hallpike's[3] cases of Ménière's disease, why was positional nystagmus, which Dix and Hallpike[8] claim to be utricular in origin not present in a similar proportion of their cases of Ménière's syndrome? In the clinical case presented, with utricular disease described histologically to account for the symptoms, there may be some argument that the dizziness and deafness could have been more casually related to the glioma and the associated central nervous system disturbance than Dix and Hallpike estimated.

From the experiments already referred to viz. those of Versteegh,[38] Tait and McNally,[37] deKleyn and Lund,[6] Graybiel, Noven and Walsh[20] there is no evidence to prove the assumption that utricular disease is associated with post-caloric nystagmus, post-rotational nystagmus, with positional nystagmus or with directional preponderance.

It would seem that as yet the origin of positional nystagmus has not been sufficiently well localized to enable Dix, and Hallpike[8] to set up a new clinical classification of cases of dizziness under that heading, or to ascribe the cause of the symptoms to utricular disease.

Notwithstanding the fact that there are still cases of dizziness which are difficult to classify, the diagnosis of Ménière's syndrome can now be established with greater assurance than ever before. This is largely due to the work of Hallpike and his associates. Hallpike's contributions to the pathology, the symptomatology, and to the differential diagnosis of Ménière's syndrome will rank with the highest achievements in otology.

MEDICAL TREATMENT

The medical treatment of dizziness is largely symptomatic because the basic cause has been so elusive. Surgical treatment, for the most part destructive, should be reserved until medical treatment has been given every chance to succeed.

When assessing the results of medical treatment one must recognize that a treatment ordered is not necessarily a treatment received

by the patient. One must be sure that the treatment was followed as outlined, that the dosage of any drug was adequate, and that it was taken over a long enough period of time.

There are certain general considerations which may be important. The patient's surroundings may be contributing or may even be causative factors. Treatment has less chance of success if the patient is under extraordinary mental strain or if he is excessively fatigued. Irregularities in habits should be corrected. Excessive smoking or excessive alcohol intake should be controlled. The history and the physical findings should determine whether or not a complete allergic investigation is necessary. If any sensitivities are found, appropriate treatment should be instituted.

Sedatives. Sedatives were, for a long time, the mainstay of medical treatment. A difficulty is that there is no such thing as a satisfactory sedative for a chronic illness such as dizziness. Experiments carried out during the war years have shown that of all the sedatives, hyoscine or some of its compounds would seem to be the most satisfactory drug for controlling labyrinthine sensitivity. The British, American, and Canadian motion sickness remedies contained hyoscine compounds. Most of the patented motion sickness remedies also contain hyoscine components.

Antihistamines. The antihistamines have been reputed to be effective in the treatment of dizziness, but since most of these have a sedative action it has been suggested that the benefit may be due to sedation. Dramamine does have some beneficial effects. It is helpful in suppository form during the acute stages of an attack of dizziness, and for the dizziness following a fenestration operation or following a labyrinthine ablation.

Sodium Free Diet. Since the report of Furstenberg, Lashmet and Lathrop[16] upon the harmful effects of sodium, a salt free diet, or a modified salt free diet has been advised, with a view to eliminating sodium. In order to counteract the effects of any remaining sodium the patient should be given amonium chloride. Williams[39] stressed the need for giving ammonium chloride—not only to counteract the sodium, as suggested by Furstenberg et al, but because he wished to counteract a potential alkalosis. Ammonium chloride tends to produce a relative acidity. He quoted Selye as stating that excessive quantities of sodium chloride sensitize the organism to a damaging agent such as cold, while the acid salts, such as ammonium chloride, tend to prevent such sensitization. Williams also claimed that ammonium chloride has a beneficial diuretic effect in Ménière's syndrome.

Vaso-dilators. Since the pathological studies of the labyrinth in Ménière's syndrome would indicate increased intra-labyrinthine pressure and fluid imbalance, the treatment has been directed towards the alleviation of a theoretical autonomic imbalance and vascular disturbance.

Two drugs in common use can both be classed as vasodilators. They are nicotinic acid and histamine. Nicotinic acid can be given by mouth, subcutaneously or even intravenously. When given in large enough doses it does have a beneficial effect upon the symptoms. The histamine may be given subcutaneously or intravenously in increasing doses. Williams[39] questioned whether the benefits of histamine depend upon its histamine desensitizing action or upon its vasodilator effect. These drugs may be given together or separately. One advantage of the nicotinic acid is that it can be given by mouth, and can be carried on over long periods of time.

Because he thought the crisis to be neurovascular, Hilger[23] recommended a neural block with intravenous procaine.

Streptomycin. Glorig and Fowler[18] suggested that toxic doses of streptomycin, given over a short period of time might be used as a treatment of vertigo. Their reasoning was that the labyrinths would be knocked out without causing interference with hearing. This treatment has received some general adoption. However, there is need for caution because there is evidence to show that the drug affects the vestibular nuclei. It is not definitely known that it has no other harmful actions. Furthermore, it destroys both labyrinths, and a patient with loss of both labyrinths is more seriously handicapped than a patient who has lost only one labyrinth. In a sensitive patient the hearing may be harmfully affected.

SURGICAL TREATMENT

Surgery should be reserved for those patients who have had a thorough and complete trial of the accepted medical treatments, and who, in spite of such trials, are inconvenienced to the point where they become handicapped. Cases of Ménière's syndrome may involve one or both ears. This is important to remember when one is considering surgical treatment. If only one ear is involved presumably the hearing in the opposite ear is normal. As a rule the hearing in a case of Ménière's syndrome, considered for surgical treatment, is below the serviceable level.

Labyrinthine Ablation. The most suitable surgical procedure is generally considered to be a labyrinthine ablation performed according to the method of Cawthorne[2] through a modified simple mastoid

operation approach. An opening is made into the labyrinth and the membranous labyrinth is exttracted by gentle traction. Day[5] who had advocated a partial labyrinthine ablation by electro-coagulation, in the hope of preserving the residual hearing, has recently advocated complete destruction of the labyrinth according to the method of Cawthorne.

Eighth Nerve Section. Section of the eighth nerve — either partial or complete—and either bilateral or unilateral may be done for relief of dizziness. Unilateral eighth nerve section would have the same effect as labyrinthine ablation, but it is a more serious surgical procedure because it is an intracranial operation. Nerve section is definitely worthy of consideration when one is faced with an intractable dizziness and when there is only partial reduction of hearing in each ear. In such a case everything must be done to protect the residual hearing in both ears, while at the same time attempting to stop the disturbing attacks of dizziness. A bilateral partial eighth nerve section can be carried out. The vestibular portion of each eighth nerve is cut, leaving the cochlear portion of the nerve intact. Dandy[4] reported the largest of any series of eighth nerve sections. He had operated upon four hundred and one patients. Green and Douglas[21] reviewed the post-operative results of this series of patients. One hundred and fifty patients were re-examined and two hundred and fifty-eight others answered a questionnaire. Twenty-three patients had been submitted to differential section of the vestibular nerve in the more affected ear. Thirteen of the twenty-three have remained free from dizziness. Dizziness was relieved in ninety percent of the whole series—whether the section was partial or complete. It should be noted that after partial nerve section, the hearing slowly declined in about forty-eight percent of the cases. This would mean that even after performing partial nerve section one is still faced with a serious problem, when trying to save the residual hearing. Even in Dandy's experienced hands facial paralysis occurred in twenty-five percent of the cases, being permanent in ten percent.

Sympathectomy. In the treatment of Ménière's syndrome, cervical sympathectomy has been advocated from time to time. Passe and Seymour[33] reported the results of injecting the cervical sympathetic ganglia and of sectioning the nerves to and from these ganglia. They reported improved hearing in many cases, cessation of dizziness in most, and relief of tinnitus in some cases. These beneficial effects, of course, are offset by the symptoms which accompany the cervical sympathectomy, the chief of which is a Horner's syndrome. This treatment is worthy of consideration because the internal ear struc-

tures are preserved and apparently, to some extent, their function may be restored.

ASSESSMENT

In assessing the value of treatment one must take into consideration the fact that frequent and long remissions are characteristic of dizziness, whatever the cause. If treatment is under way when a remission sets in, that treatment may be given credit for the so-called cure. McNally, Stuart, Lockhart and McKercher[29] found that remissions averaged about four years. They, therefore, suggested that the results of any treatment should not be considered a cure unless the patient has been symptom free for at least five years.

SUMMARY

If a patient complains of dizziness, one must first exclude the possibility that it is a warning sign of imminent labyrinthine invasion in the course of suppurative middle ear disease, either quiescent or active. Fistula and other vestibular tests should be performed at once.

Next, evidence should be sought for serious general organic disease, such as cardio-vascular or central nervous system lesions which were found as often as in thirty-five percent of a consecutive series of two hundred cases of dizziness.

True Ménière's syndrome or labyrinthine hydrops will be the diagnosis in about thirty percent of the cases. This diagnosis can now be more firmly established because of the cochlear signs of recruitment, lowered threshold of discomfort and reduced intelligibility at higher intensities.

The remaining thirty-five percent of the cases, those in which the dizziness is not associated with cochlear signs or symptoms, are still difficult diagnostic problems and can best be grouped as atypical or pseudo-Ménière's syndrome, constant watch being kept for other diagnostic signs.

The origin of positional nystagmus or of directional preponderance is not yet well enough established for these signs to have any great diagnostic significance.

The treatment of dizziness is still, for the most part, symptomatic. A case of Ménière's syndrome should be given a thorough trial of medical treatment before surgery is contemplated.

Hyosine and its compounds have proven to be among the most effective labyrinthine sedatives. Antihistaminic preparations have

been helpful and it is thought to be because of their sedative properties.

Because of a prevalent theory that labyrinthine hydrops is the result of vascular spasm, vaso-dilator drugs play an important role in treatment. Nicotinic acid and histamine are the two in most common use.

The operation of choice for Ménière's syndrome, when the disease is unilateral, is labyrinthine ablation. If the disease is bilateral a differential section of the vestibular portions of both eighth nerves should be considered, in the hope of preserving some of the residual hearing.

1509 Sherbrooke St. W.

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LVI

ABSORPTION FROM NASAL MUCOUS MEMBRANE: SYSTEMIC EFFECT OF HYOSCINE FOLLOWING INTRANASAL ADMINISTRATION

JUERGEN TONNDORF, M.D.

REED W. HYDE, CAPT. USAF (MC)

HERMAN I. CHINN, PH.D.

AND

JAMES E. LETT, LT. COL. USAF (MC)

RANDOLPH FIELD, TEXAS

A number of drugs administered prior to exposure have been shown to afford significant protection against motion sickness.[2] The value of these drugs in curing the condition, however, once it has been induced has not been clearly defined. For a preparation to be of maximum value therapeutically, it should be rapidly absorbed and easily administered by the patient himself. Oral administration to a nauseated or vomiting patient has the obvious disadvantages of difficulty in swallowing and retaining the drug and a relatively slow rate of absorption. Similarly, self medication by rectum and by injection are impractical. The intranasal route would seem to be ideally suited for this purpose because of the vascularity of this site and the ease of administration. Unfortunately, reports on absorption by the nasal mucosa are conflicting. The rapid systemic response to cocaine "snuffs" is a matter of common knowledge. Yoffey[10] was able to detect trypan blue in the cervical lymph tract within 20 to 30 minutes after its instillation in a monkey's nose. Further, Clark[3] found potassium ferrocyanide and ferric ammonium citrate to have penetrated into the cranial cavity within an hour after nasal administration.

In contrast to these findings are those by Childrey and Essex[1] who found no apparent systemic effect in dogs after instillation of various drugs (antihistaminics, adrenalin and its derivatives, cocaine, nicotine) into the frontal sinus which had been sealed off from the nasal cavity. In experiments on rabbits, Gundrum[4] also detected

no systemic side effects when antihistamines were given intranasally. After nasal instillation of adrenalin derivatives in human subjects, Van Alyea[9] noted negligible systemic effects. He attributes the systemic effects which are sometimes observed following intranasal medication largely to swallowing of excess fluid by the subject. Therefore, he warns emphatically against swallowing any of the excess fluid which may spill over from the nose.

Before testing the efficacy of anti-motion sickness preparations instilled in this manner, it seemed necessary, therefore, to investigate further the rate of absorption and the conditions required for optimum action. The relatively high dosage required for protection by most preparations precluded their use in this connection. Hyoscine (scopolamine hydrobromide) by oral application, however, requires only 0.65 mgm for protection, a quantity easily administered intranasally. For this reason, hyoscine was the test drug employed throughout. The rapidity and degree of activity of hyoscine administered orally, nasally, and subcutaneously were compared.

EXPERIMENTS

Since methods were not available for the chemical determination of hyoscine in body fluids, the quantity absorbed was estimated by the pharmacological response induced. The most obvious effects of hyoscine in moderate amounts are pupillary dilation, cycloplegia, and drying of the mouth. No consistent ophthalmic changes could be detected with the dosage employed, however, so that, on suggestion of one of the authors (Chinn), the decreased salivary flow was utilized in all experiments to be described.

The subjects were Air Force recruits, 17 to 20 years old, and in apparently good health. Each man receiving nasal medication was examined for evidence of head colds. Approximately 13.5 per cent of these subjects showed swelling of the turbinates or purulent exudate or both. After a brief explanation of the test procedure, each man was given a stick of chewing gum for each test period and instructed to collect all saliva in a container of known weight for exactly five minutes. The saliva was weighed to the 0.01 gram. With but few exceptions, where only one control sample was taken, two control samples were collected fifteen minutes apart, using fresh sticks of gum each time. The hyoscine was then administered and salivary secretion measured at 15 and 30-minute intervals up to a total period of two hours; the results were expressed as percentage values of the control. All changes in saliva production described in this paper have probability values of 0.05 or better unless stated otherwise.

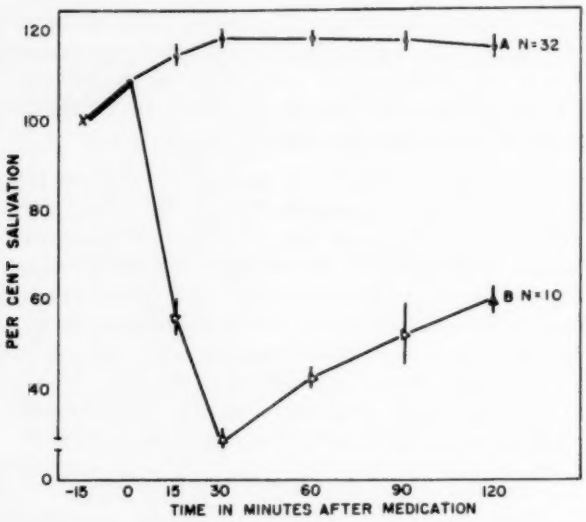

Fig. 1.—Change in saliva production expressed as per cent of first control speciman (−15). Curve A—non-medicated controls; Curve B—after subcutaneous injection of 0.65 mgm hyoscine. Plotted are means and one standard error (vertical line); numbers of subjects as indicated (N).

Before studying the action of hyoscine, the constancy of salivary production during periods of up to two and one-half hours was first determined. This control group consisted of subjects receiving either no medication, 0.4 cc of 0.9 percent saline intranasally or a placebo capsule by mouth. Since no difference was evident among these subgroups, they will be considered as a single control group. It is apparent from Figure 1 that salivary production increases slightly but significantly during the first hour, after which it remains at a constant level for the duration of the experiment.

In the next experiment, hyoscine (0.65 mgm) was given subcutaneously (Fig. 1). The absorption was rapid, as indicated by a diminution of saliva within 15 minutes ($P<.001$). The peak of the effect was reached after 30 minutes, followed by a gradual return towards normal production. The salivary flow at each interval measured was markedly lower than the control value at a corresponding period ($P \ll 0.01$).

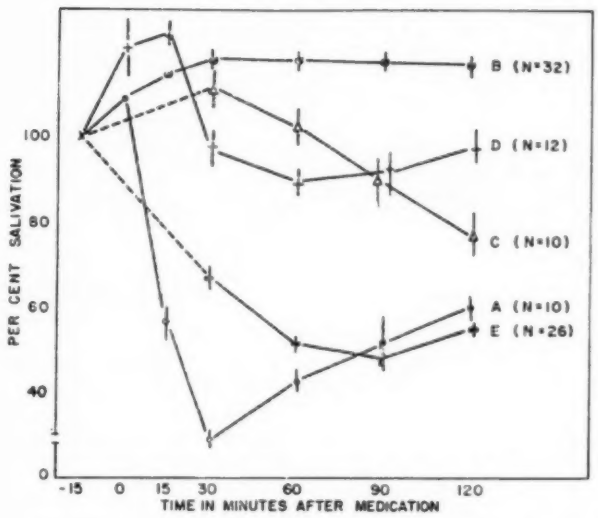

Fig. 2.—Change in saliva production with time expressed as per cent of first control specimen (−15) after medication with 0.65 mgm hyoscine. Curve A—subcutaneous injection; Curve B—non-medicated controls; Curve C—oral capsules; Curve D—oral fluid; Curve E—nose drops. Plotted are means and one standard error; numbers of subjects as indicated.

Oral administration consisted of 0.35, 0.65, and 1.0 mgm of hyoscine in identical No. 1 pink capsules. In a single series, 0.65 mgm was given in 20 ml of water rather than in capsule form. For nasal application, 0.2 ml of varying concentrations in saline (pH 5.7-6.0) was instilled in each nostril under direct visual control, with the aid of a blunt needle and a small calibrated syringe. Larger quantities were found to spill down the throat.

The importance of the route of administration is clearly indicated by Figure 2, which compares the anti-sialologic action of hyoscine by oral, nasal, and subcutaneous administration. In all cases, salivary production was significantly reduced below control levels, followed by a gradual return toward normal levels. Capsules taken orally gave the slowest response, followed by oral liquid ingestion. The delay in the former case apparently depends upon the time required to dissolve the capsule. Subcutaneous injection gave the most marked and rapid response and nasal instillation occupied an intermediate position.

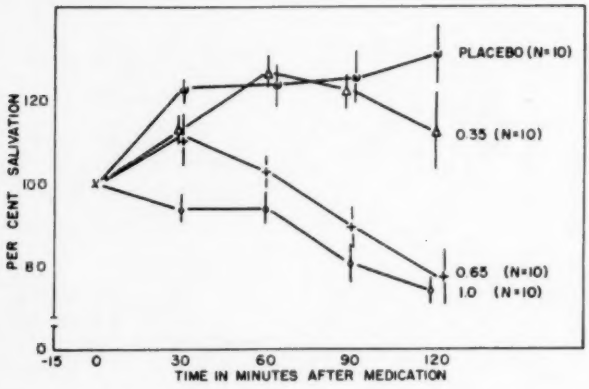

Fig. 3.—Change in saliva production with time after oral medication (capsules). Dosages and number of subjects as indicated.

The effect of dosage variations orally and nasally are shown in Figures 3 and 4. The greater action of the drug given by nose compared to that by mouth is apparent. A dose of 0.35 mgm by mouth was completely ineffective, whereas 0.4 mgm by nose markedly reduced the salivary flow. On oral application, dosages of 0.65 and 1.0 mgm were clearly effective, although statistically there was no distinction in the effect between these two latter dosages. The effect of the lower dosages (0.1 to 0.4 mgm) of nasal medication could not be differentiated, while clear separation was made between the 0.4, the 0.6, and the 1.0 mgm dosages.

The possibility that the swelling of the nasal mucous membrane, as observed in some cases, governed the rate of hyoscine absorption prompted study of neosynephrine action. The cavernous tissue of the turbinates was temporarily shrunken by administration of phenylephrine hydrochloride (neosynephrine) nose drops (0.2 ml of 0.25 per cent solution in each nostril). In one group, the drops were given 15 minutes and in another immediately before hyoscine administration. Figure 5 shows the result of two different dosages of hyoscine (0.3 and 0.6 mgm) with neosynephrine as well as corresponding curves without neosynephrine. Neosynephrine hindered the action of 0.6 mgm hyoscine considerably when given immediately before the drug ($P<0.01$) but merely delayed the action when

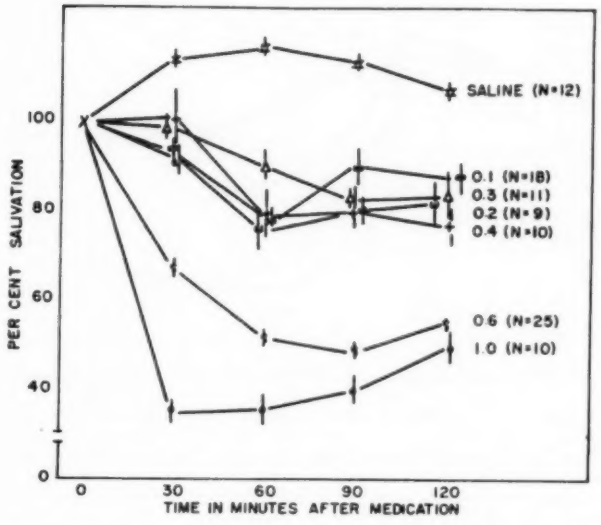

Fig. 4—Change in saliva production with time after application as nose drops.

given 15 minutes before. The difference in the two curves at the 0.3 mgm dosage level indicates a similar trend although it is too small for statistical significance.

For routine use, application of a nasal spray affords a simpler technique than the instillation of nose drops. Using plastic spray bottles, each squirt sprayed 0.025 grams of fluid with an in-bottle standard deviation of 0.011 grams and an average between-bottle standard deviation of 0.012 grams. The required dosage of 0.6 mgm of hyoscine was contained in 0.2 cc, or roughly equivalent to six squirts. Three squirts were applied to each nostril. The exact amount of fluid used in each case was determined by weighing the bottles before and after use.

The subjects in this series received an average of 0.286 cc per dose or 0.85 mgm of hyoscine. Despite this larger dose, the subjects showed significantly less response than those receiving 0.6 mgm as nose drops (Fig. 6). Furthermore, in the spray group, dependence of the drug effect upon the actual amount of hyoscine used was not high. Coefficients of correlation at the varying time intervals ranged from -0.38 to $+0.06$.

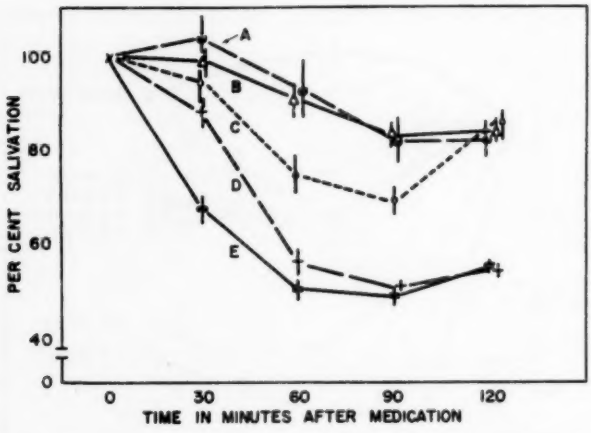

Fig. 5.—Effect of neosynephrine upon the change in saliva production with time after intranasal medication with hyoscine: hyoscine dosages; 0.3 and 0.6 mg. Curve A; neosynephrine 15 minutes before 0.3 mg hyoscine. Curve B; 0.3 mg hyoscine with no neosynephrine. Curve C; neosynephrine immediately before 0.6 mg hyoscine. Curve D; neosynephrine 15 minutes before 0.6 mg hyoscine. Curve E; 0.6 mg hyoscine with no neosynephrine.

The lowered hyoscine effect of nasal spray compared with nose-drop instillation under controlled conditions was somewhat surprising. It is known,[7] however, that the entire lining of the nose is covered by a thin but rather viscous coat of mucus. Hence, it is not unlikely that, because of the relation of droplet size to surface tension, the fine droplets produced by the spray are confined to the spot at which they impinge, thus resulting in poor "wetting" of the epithelium. Part of the drug may even be swept down into the nasopharynx by the movement of the mucous film (approx. 2.5-7.5 mm/minute) and thus remain practically ineffective. The comparatively large drops dispensed by the syringe, on the other hand, may spread to much wider areas and establish better contact with the epithelium.

If this explanation is correct, the addition of a wetting agent might lower the surface tension of the endonasal mucous film and permit better absorption of the drug. Solutions of the detergent, sodium lauryl sulfate (Duponal C), in saline were tested subjectively for their irritation to nasal mucosa. A few drops of a 0.005 per cent solution produced no sensation, instillation of 0.05 per cent so-

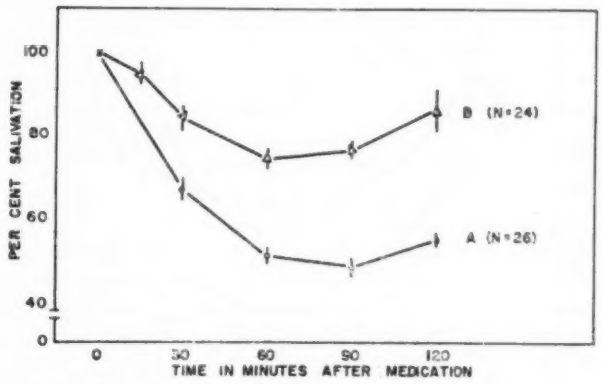

Fig. 6.—Change in saliva production with time after intranasal medication with hyoscine. Curve A—nose drops (0.6 mgm); Curve B—spray (actual average dose: 0.85 mgm).

lution caused slight burning, and of 0.5 per cent solution a marked burning. Since Proetz[7] had reported that 0.01 percent of detergent caused no perceptible damage to living mucosa and since this concentration still retained marked surface activity, solutions of hyoscine were prepared in 0.01 percent Duponal-saline solution and administered both as drops and as spray. The pH of the solution was not affected by this concentration.

As is evident in Figure 7, the nose drops containing detergent allowed a slightly more rapid absorption of hyoscine compared with the unfortified nose drops. The addition of detergent to the spray increased strikingly the degree of hyoscine absorption. Despite the administration of a slightly lower average dose (0.70 vs. 0.82 mgm) the salivary flow was consistently lessened with the duponal solution as compared to that without the detergent ($P<0.01$) and even more than in the nose drop series for which the 0.60 mgm dosage had been used. Thus, the addition of detergent to the nasal hyoscine spray provided as effective a medium of drug administration as nose drops.

COMMENT

Since the experiments using subcutaneous injection represent the baseline for all other tests, their results were investigated from another aspect. In order to check how closely the single individual followed the average pattern, presented in Figure 1, the coefficient

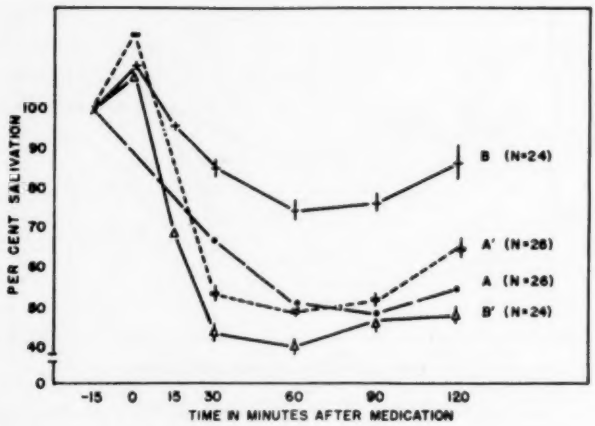

Fig. 7.—Effect of detergent on nasal absorption of hyoscine expressed by the change in saliva production with time. Curve A—nose drops without detergent; Curve A'—nose drops with detergent; Curve B—spray without detergent (actual average dosage: 0.85 mgm); Curve B'—spray with detergent (actual average dosage: 0.70 mgm).

of correlation was established between the zero control and the 30-minute point and also between the latter and the 120-minute point. As indicated by the results (1.0 and 0.78 respectively), individuals correlate quite well with the average.

In addition, comparison was made of the standard deviation at each check point between this and the control series. At the peak level of the hyoscine effect (30-minute point), it was found significantly lower than in the control group in which it was fairly stable over the total time of observation. This finding was to be expected: If the action of a drug tends to approach a saturation value, i.e., zero percent salivation, the variance should decrease accordingly. Thus, the degree of variance at the peak level of the hyoscine effect may be taken as an index of the uniformity of drug action. In line with this finding, a comparison was made of the various forms of application. Using subcutaneous injection as the reference, the uniformity of drug action was found quite unsatisfactory with oral application, but fairly good with nose drops. Addition of detergent improved it further, although the difference was too small for statistical significance. In the nasal spray series, uniformity was again not too good, but was markedly improved after addition of the detergent.

These findings demonstrate the utility of nasal application for drug administration. There can be little doubt that the absorption of hyoscine through the nasal mucosa occurs with a speed and completeness more nearly resembling subcutaneous than oral administration. Our findings are not surprising in view of the structural differences in the mucous membrane involved. The blood supply is abundant for the nasal mucosa, particularly within the turbinates, less so within the digestive tract, and rather scanty within the nasal sinuses. This is at variance with those reports that little systemic effect resulted through this route after instillation of antihistaminic and adrenalin-like substances and that such effects were caused by swallowing of excess fluid. However, differences in drug action may partly reconcile these earlier findings with those reported herein. The drugs used in the earlier experiments were strong vasoconstrictors acting upon the cavernous tissue within the nose. Thus, they hindered their own absorption into the systemic circulation. Supporting evidence for this concept is the observation that simultaneous application of neosynephrine decreased the activity of hyoscine.

No data are available to explain the gradual increase of salivary production in the non-medicated control. It may be the result of a simple training process whereby less saliva is swallowed, allowing a more efficient collection. Whatever the explanation, it renders the negative slope of the medicated groups all the more significant.

One point remains to be discussed: That is whether hyoscine or Duponal or both together, may or may not exert a local noxious effect upon the nasal tissues. The sensitivity of the nasal ciliated epithelium to some agents such as cocaine and adrenalin is well known.[7] On the other hand, it is also known that the pain threshold of the nasal mucous membrane is quite low. All our subjects, a total of 223 individuals, were asked whether they noticed any burning sensation or pain in the nose after medication. All of them reported in the negative.

Furthermore, scopolamine is applied routinely to another sensitive mucous membrane—the conjunctiva of the eye—with very rare ill effects, according to clinical experience. One drop of a 0.25 per cent solution is used; this gives an approximate dosage of 0.5 mgm which is comparable to the dosages used in the present experiments. Less than 0.1 per cent of the patients may develop a conjunctivitis which is thought to be caused by individual hypersensitivity.[11] We may infer from this observation, therefore, that a single application of hyoscine in the dose range from 0.1 to 1.0 mgm has no detrimental

effect upon the nasal mucosa. Repeated intranasal use of the drug may lead in some individuals to the development of a hypersensitivity; this question remains to be evaluated.

Detergents have recently been employed with considerable success for the purpose of liquefying bronchial secretions, especially in small children.[6, 8] Based on clinical experience as well as animal experimentation, the absence of toxic reaction was emphasized. As a safe upper limit of concentration of the detergent, a value of 0.1 per cent was given.[5] Although the dosage used in the present experiments (0.01 per cent) appears rather conservative, additional clinical and experimental studies are indicated.

SUMMARY

1. Intranasal medication with hyoscine as drops and as sprays was compared with oral and subcutaneous administration. Using the relative decrease in saliva production as an indication, hyoscine was shown to be more rapidly, completely, and uniformly absorbed intranasally than orally. Drops were superior to the spray.

2. The addition of a detergent to the hyoscine spray markedly improved its activity, presumably by allowing penetration of the mucous layer.

3. Neosynephrine given intranasally before hyoscine administration decreased the hyoscine effect.

4. The action of vasoconstrictors in hindering absorption is discussed.

School of Aviation Medicine.

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LVII

HEREDITARY HEMORRHAGIC TELANGIECTASIA

DAVID A. DOLOWITZ, M.D.

O. N. RAMBO, JR., M.D.

AND

F. E. STEPHENS, PH.D.

SALT LAKE CITY, UTAH

Hereditary hemorrhagic telangiectasia is a vascular disease characterized by dilatation of arterioles, capillaries and venules into angiomata. These lesions are hereditary and the ectatic vessels frequently bleed spontaneously or after the slightest trauma. Although these lesions appear in all parts of the body, one of the most common sites is the oronasal cavity. Since nasal lesions are especially susceptible to injury, severe epistaxis frequently brings patients with this disease to the otolaryngologist. This offers him an opportunity to make a definitive diagnosis and to initiate studies to determine whether similar lesions exist elsewhere in the body.

The State of Utah offers a unique opportunity to study hereditary conditions. The population is agricultural in a large measure, and since the farms are self-owned, the people tend to remain in the same vicinities. Polygamy among the early settlers has resulted in large kindreds. These conditions, coupled with the availability of geneological records kept by the Mormon Church greatly facilitate genetic investigations.

This is a study of a kindred in which hereditary hemorrhagic telangiectasia has occurred in the past six generations. After the diagnosis had been established in several members of the family, interest was aroused in studying the condition.

From the Departments of Surgery, Medicine and Pathology, the Laboratory for the Study of Hereditary and Metabolic Disorders, University of Utah College of Medicine, and the Laboratory Service of the Veterans Administration Hospital, Salt Lake City, Utah.

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Presented to the Thirty-seventh Annual Meeting of the Pacific Coast Oto-ophthalmological Society, Tuesday, May 26, 1953.

Methods: A team, composed of an internist, an otolaryngologist and a geneticist, visited key homes where members of the families congregated. Detailed histories were taken on each individual. If the individual was not available, data were used only when verified by three independent sources.

Examination was limited to the areas of telangiectasia. The appearances of lesions and clots were recorded. Various bleeding and clotting studies were then made. These data were supplemented by histories, physical examinations and laboratory studies of those members of the kindred who were patients at the Salt Lake County General Hospital or the Veterans Administration Hospital at Salt Lake City or under office care for epistaxis.

Genetic Studies: At the time of writing data were available concerning 491 descendants of an early pioneer. One hundred ninety-four individuals were seen and examined. There was a history of bleeding in at least eighty-three cases. Thirty-eight of these bleeders were examined and the diagnosis of telangiectasia with hemorrhage was confirmed in them.

Figure 1 shows a summary of the appearance of the familial trait. The squares represent men, the circles women, and the smaller symbols children. Open symbols indicate normalcy, solid black, telangiectasia, and cross-hatched, inadequate information.

The progenitor designated as I-1 came to Utah from New England. He had four wives. Since the trait descended through him, his progeny by all wives were considered as one group, Generation II.

This second generation consisted of twenty-two members, nine men and thirteen women. The bleeding tendency was reported in nine of this group, five men and four women.

The third generation was composed of their 143 offspring. Twelve men and thirteen women are known to have carried the trait. It was reported that another male also was afflicted with nasal hemorrhage, but this could not be cross checked and the data were not accepted.

Generation IV was the most difficult to trace. Adequate data were obtained concerning 154 members but the information concerning thirty-eight additional siblings was not sufficiently complete to permit its use. The trait appeared in thirty-two of the acceptable cases, affecting sixteen members of each sex.

The fifth generation is composed of 127 members. In twenty-six cases the data had to be disregarded for lack of adequate cross

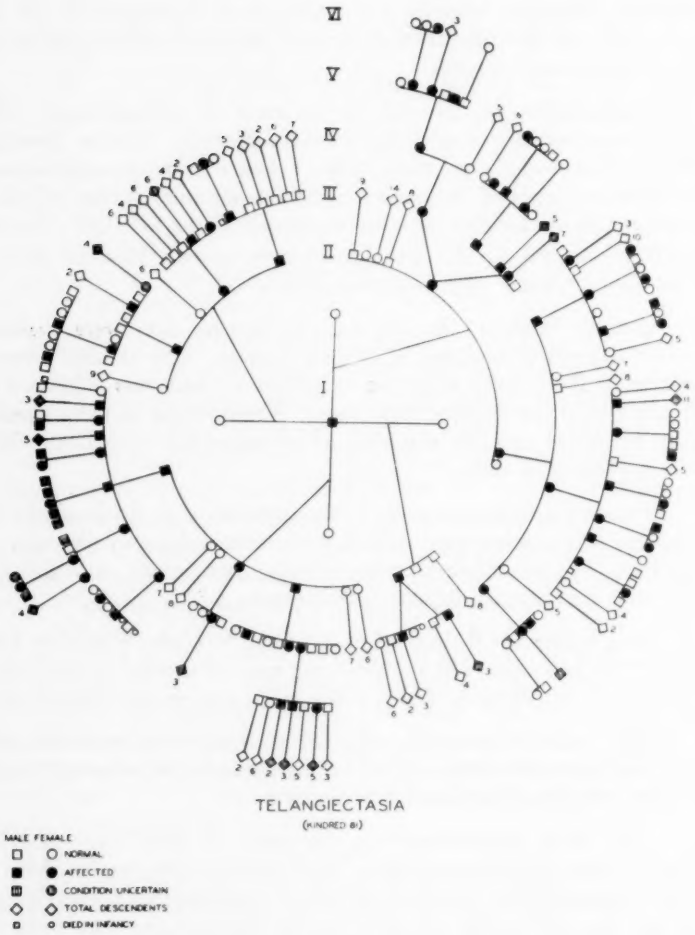


Fig. 1.—Schema indicating incidence of telangiectasia in the kindred under review.

checking. Ten women and five men already show bleeding. At the time of writing, the sixth generation consists of seven children. So far, only one girl is afflicted.

Study of this group shows that the trait is always present in one parent of an affected individual and is inherited as a Mendelian dominant. Since penetrance is complete, only 197 members of this kindred are capable of receiving the trait, ninety-four males and 103 females. Actually, thirty-nine men and forty-four women had developed telangiectatic lesions, representing an equal genetic division between the sexes.

In this kindred there have been no marriages of two telangiectatic individuals. When a mating occurs between a normal individual and one with a dominant trait, equal numbers of normal and afflicted children are expected. In those families of this kindred where a parent was afflicted and the data were sufficiently complete to be accurate, a ratio of eighty-three afflicted to eighty-seven normals was secured, substantially the expected one to one ratio.

Snyder and Doan[1] report a mating of two parents with hereditary hemorrhagic telangiectasia. The offspring was presumably homozygous. At birth there was a large port wine stain on the infant's chest. Almost immediately the telangiectasia began to appear. The baby died in less than three months. At autopsy the skin and many organs were involved by telangiectatic lesions. It would seem that telangiectasia is lethal in the homozygous state.

Clinical Observations: Grossly, the lesions appeared as dilated vessels, varying in color from red to purple and in size from pinpoint to large spider-like formations (Fig. 2 and 3). Usually these lesions blanched on pressure.

The age of onset varied considerably. Usually the first sign was nosebleed occurring in childhood. The earliest occurred at three months, the latest in a man of sixty. About puberty, in most cases, the bleeding tendency disappeared. In the thirties the dilated vessels began to appear on the skin or mucous membranes followed soon afterward by hemorrhage from these telangiectatic lesions.

Hemorrhage was the chief complaint of this disease. The incidence of mortality is unknown. In eight cases of this kindred external bleeding was sufficiently profuse to cause death. If hemorrhage is prevented or controlled, this disease is not inconsistent wit longevity.

Fig. 2.—Telangiectatic lesions of the face which were not apparent at autopsy.

As in most reported families, the most common site of hemorrhage was the nose. Epistaxis occurred in sixty-eight of the eighty-three bleeders. Lesions were found frequently in the mucous membranes of the nose, mouth, and lips, in the conjuctiva and in the skin of the face, fingers and toes. One woman had a scalp that bled frequently. Finally, combing her hair started an uncontrollable hemorrhage that caused her demise.

Several members died following internal bleeding, cerebral, pulmonary or gastro-intestinal. In the absence of an autopsy, telangiectasia can only be suspected as a cause of these hemorrhages. The literature shows reports of lesions in virtually all parts of the body, including the urinary tract,[2] stomach,[3, 6] tracheo-bronchial tree,[4] uterus,[5] rectum,[1] brain[7] and lungs.[8] Moyer and Ackerman[8] describe removal of arteriovenous fitulae associated with extensive pulmonary telangiectasia in three members of a family. Basing their opinions on this finding, they postulate hereditary localization.

Fig. 3—Telangiectatic lesions of the face which were not apparent at autopsy.

There have been several deaths following internal bleeding. In this series, the only patient in whom this complication was investigated showed a bleeding ulcer at operation. Because of the paucity of clinical studies, the effect of this disease on internal organs can best be observed at autopsy.

Pathologic Studies: Review of the literature reveals that very little has been added to the brief description in Osler's paper of 1901.[14] "Section of the nasal septum . . . showed many large dilated veins just beneath the epithelium."

There are several reasons for this deficiency of knowledge of the pathologic anatomy of hereditary telangiectasia. The lesions are analogous to esophageal varices, which at death may collapse so completely that they are overlooked by the pathologist (see Fig. 2) unless he uses special injection methods to demonstrate them. Histologically, the ectasias are units of unique simplicity, possessing little more than a layer of delicate endothelium. Another barrier to complete study

is the location of the majority of the clinically manifest lesions. Routine autopsy technics rarely include examination of nasal mucosa, tongue or pharynx. Excision of facial lesions is scrupulously avoided. Biopsies have proved to be the most valuable source of material.

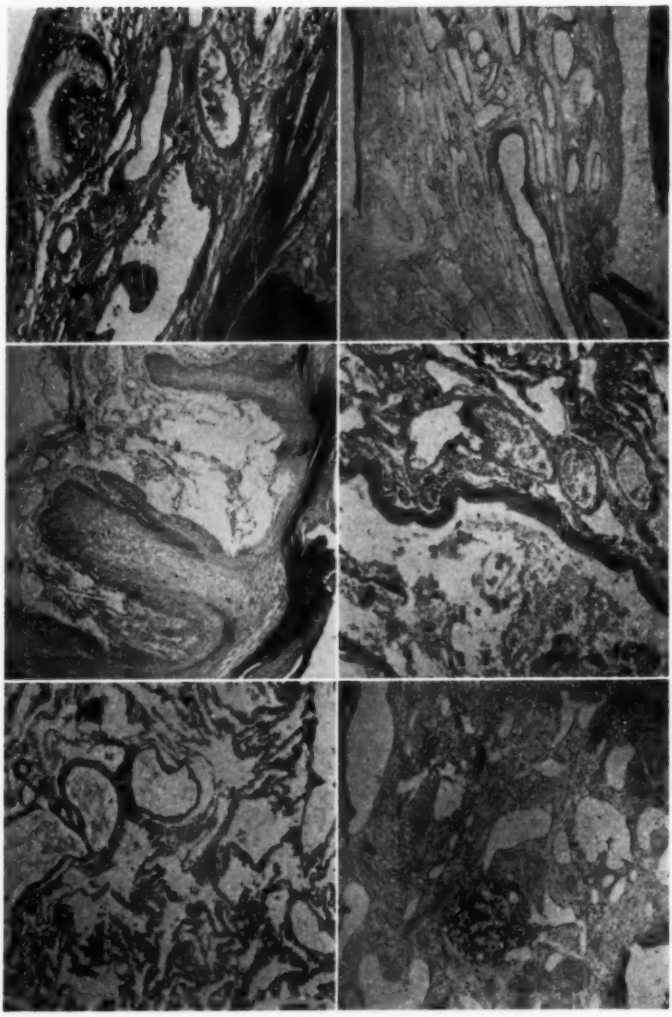
In addition to the basic lesion, which is a greatly dilated endothelial space devoid of elastic and muscle fibre, other changes must be regarded as complications of mechanical stretching, rupture and hemorrhage. These include thrombosis with obliteration of telangiectases, "degenerative" changes in supporting stroma with disintegration of connective tissue and splitting and disappearance of elastic fibers and "proliferative" phenomena, such as vascular budding.

One aspect of the condition that has not been sufficiently stressed or investigated is the fact that it may be generalized. The known hereditary nature of the disease and the generally accepted concept that it is a defect in capillary walls would make local selectivity of site difficult to explain. The difficulty is increased by the multiplicity of sites that have been reported. It is postulated that susceptibility to trauma determines the site of hemorrhage. The greater incidence of nasal bleeding in arid climates supports this theory.

In one of our cases (B.E.B., AV-296) (see Fig. 3), the chief clinical manifestation was epistaxis. Cutaneous lesions were also present and there were telangiectases of the buccal mucosa. Autopsy showed definite lesions in the nasal and buccal mucosa and revealed identical dilated thin-walled endothelial spaces in the posterior pituitary, pulmonary alveoli, subepithelial zone of the bronchi, adrenal capsule and peritruncal zones of the liver and spleen (see Fig. 4). There were questionable lesions in the bowel, epicardium and parathyroids. The possibility of overdiagnosis is appreciated; the difference between true lesions and dilated capillaries is subtle.

Further pathologic investigation of this condition can be greatly helped by a more aggressive attitude on the part of clinicians. More biopsies should be obtained. The pathologist should be guided in deviating from his autopsy routine. Close cooperation with the clinician will enable the pathologist to find lesions that collapse and disappear at death. If a number of clinically evident sites of lesions are examined, disseminated silent lesions, protected from trauma, will be more accurately evaluated and not dismissed as "dilated capillaries."

The abnormal laboratory findings can all be accounted for by severe hemorrhage. Bleeding and clotting studies, tourniquet tests and platelet counts all showed normal results. White blood counts,



TURBINATE
TONGUE
LUNG
NASAL SEPTUM
BRONCHUS
PITUITARY

Fig. 4.—Photomicrographs of telangiectatic lesions in various parts of the body.

blood cultures, serologic and tuberculin tests provided no evidence that infection played any role. Blood chemistry studies, including icteric indices and liver function tests, were all normal. Even circulation times were within normal limits. These results, combined with the pathologic findings, indicate that telangiectasia is a hereditary disease of blood vessels and not of the clotting mechanism.

Therapy: Therapy for the disease can be divided into prevention of hemorrhage and its arrest once started. Prophylaxis has been most unsatisfactory. The literature contains many reports of methods which have been used to eradicate the lesions. Chromic acid or phenol applications topically and local injections of rutin, Vitamin K, Koagamen or snake venom, x-ray or radium have all been tried, unsuccessfully.

Although it is easy to destroy the primary lesion, satellite lesions soon form several millimeters away. Hormone therapy with conjugated estrogens has failed, despite pushing its use until pseudolactation resulted in one man. Because of a recent report of successful therapy with ethinyl estradiol[9] however, the effect of this agent is now being studied in several patients.

In attempts to arrest an existing hemorrhage, the excessive fragility of the telangiectatic vessels must be recognized. If care is exercised, oxycel, gelfoam or similar topical hemostatic agents can usually be depended upon to control the hemorrhage. These have the added advantage that they do not have to be removed after the bleeding has stopped. The clot is not disturbed, and a secondary hemorrhage is thus frequently averted.

Hereditary hemorrhagic telangiectasia has been considered a relatively uncommon disease. Figi and Watkins[10] found only twenty cases at the Mayo Clinic during a twenty-year period. However, in 1944, Barrock[11] and Snyder and Doan,[1] working independently, found over a thousand cases had been reported in the eighty-nine years since the disease was first described. Our findings of four sporadic cases and another kindred in this locality make us feel that this disease is more prevalent than suspected.

It has been suggested that this trait may be self-limiting. The reports of Teahan[12] and Alban[13] and ourselves show kindreds that have been followed for six generations. Since telangiectasia has been recognized as an entity for less than a century, the findings would seem inconsistent with the expectations of a spontaneous disappearrance.

SUMMARY AND CONCLUSIONS

1. A genetic study of a kindred of 491 members is reported. The families were traced for six generations.

2. Eighty-three cases of hereditary hemorrhagic telangiectasia occurred in this group. Thirty-eight of these were examined.

3. This disease behaves as a Mendelian dominant.

4. Pathologic changes are confined to blood vessels and the results of hemorrhage.

5. The basic lesion is a greatly dilated endothelial space which is deficient in elastic and muscular components.

6. Although attention is usually centered on local manifestations, careful search may show the lesions to be widespread.

7. Prophylactic therapy is still generally unsuccessful. Control of hemorrhage, therefore, remains the realistic treatment.

915 East First South.

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LVIII

USE OF A SINGLE CONICAL GRAFT IN MASTOID CAVITIES

MORTIMER LEE WILLIAMS, M.D.

ROANOKE, VIRGINIA

The technique described in this paper utilizes a single split-thickness skin graft, which in our experience with 14 unselected patients with long standing chronic middle ear infection and marked impairment of hearing resulted in an asymptomatic, totally dry and completely epithelized tympanomastoid cavity. Cholesteatoma was found at operation in three of these patients, and two others had had a previous radical mastoid operation, the infection persisted and their ears were discharging.

In four patients no granulation tissue appeared after operation and the graft was a complete "take" at the end of the third week. The graft successfully sealed off from the nasopharynx the tympanic orifice of the eustachian tube in 13 of our 14 patients, and examination two months after the operated ear had completely healed showed dry grafted cavities in each of these 13 patients. In the one whose eustachian tube was not satisfactorily sealed off there was discharge in the ear whenever the patient had an upper respiratory infection. Small areas of granulations developed in the grafted cavities in all of the ten remaining patients. This delayed complete epithelization and prolonged the period of postoperative treatments to five weeks for 5 patients, seven weeks for 3 patients and ten weeks for two. No systemic or local complications developed in any of our 14 cases, with the exception of the persistent small areas of granulations. These areas were always small, and we have estimated the "take" to be better than ninety per cent in each of the grafted ears. Furthermore in no patient was there any stenosis of the external auditory canal. The hearing in some of the younger patients was temporarily improved by the removal of granulations and cure of infection, but subsequent subepithelial scar tissue formation apparently interfered with the mobility of the stapes foot plate and the hearing gradually reverted to the preoperative level. It is possible that the end result might be better

From the Otolaryngological Service of the Johns Hopkins Hospital.

if the area directly over and around the stapes were grafted with mucous membrane removed from the lower lip.

Shaping the Mastoid Cavity.—An endaural radical mastoidectomy was performed in the usual manner, care being taken to remove all infected mastoid cells. Sharp depressions or pockets produced by partially uncovered cells must be saucerized and their membranous linings removed. When necessary to remove all infected bone one should not hesitate to freely expose the dura and lateral venous sinus. If the fenestration drills are used, the polishing burrs should be avoided, since the fine dust produced tends to seal the small bony canaliculi and their nutrient vessels. A slightly roughened surface is more to be desired. When the posterior canal wall has been taken down, the next step is to remove the granulation tissue and mucous membrane from the middle ear, especially from the hypotympanum and from within the eustachian tube orifice. Only partial success is usually encountered in removing granulations in the area of the stapes and facial nerve. It is in these areas that it is most difficult to get a 100 per cent take of a split-thickness skin graft. The bony excavation is carried well anteriorly, thereby obliterating the lateral ledge of the anterior portion of the middle ear. This gives a smoother surface in this area and also facilitates thorough cleaning out of the middle ear. Some of the skin lining the external auditory canal is sacrificed, but that in the outer half of the canal anteriorly is left as a protective covering to the temporo-mandibular joint. The canal skin is often damaged by chronic suppuration and harbors infection. When the skin over the joint area looks unhealthy, it should be removed and the underlying area covered with the graft. If the joint capsule should be entered inadvertantly, the graft will not take over the defect but the joint capsule usually heals without complications.

Finally it is most important to thoroughly rinse the prepared cavity with normal saline to float away bone dust, chips and clots, and then to fill the cavity with a specific antibiotic solution and leave it there while the graft is being drawn. It might be of interest to add that two of the author's cases showed B. Proteus infection with no sensitivity to the usual antibiotics, but the end result after grafting was excellent.

Preparing the Graft.—The graft should be drawn just prior to application. A split-thickness skin graft approximately 2 x 4 inches in size and eight to ten one-thousandth of an inch in thickness is desired. It is taken from the upper thigh or lower abdomen. The latter area is a wiser choice in female patients. To prevent infection of the area from which the graft is taken, gown and gloves must be

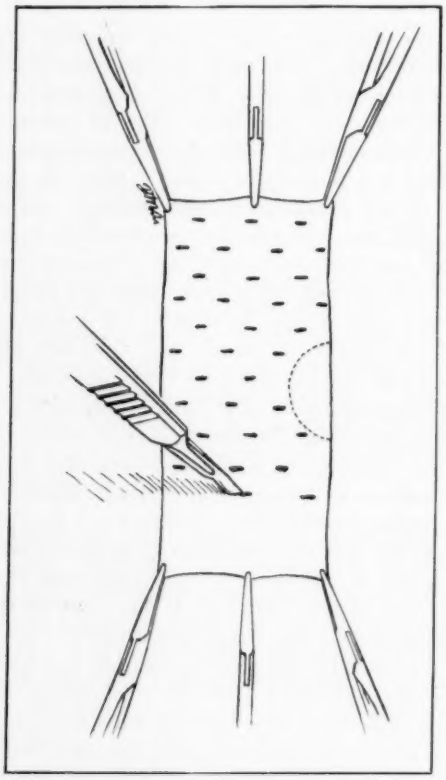

Fig. 1.—Shows the split thickness graft approximately one-half normal size; the small stab incisions ("pie crusting") are staggered leaving a small semi-circular free area in the mid portion of the graft along the straighter edge.

changed before drawing the graft. Before the surgeon returns to the mastoid field the donor site is covered with double thickness vaseline gauze and dry sterile dressings. In the meantime both the assistant and the nurse rinse their gloves and all necessary instruments in some antiseptic solution and redrape the mastoid field.

A graft of uniform thickness is obtained more readily with a dermatome. Thin grafts always give a more satisfactory take over bone. Small stab incisions and trimming of the ragged edges of the

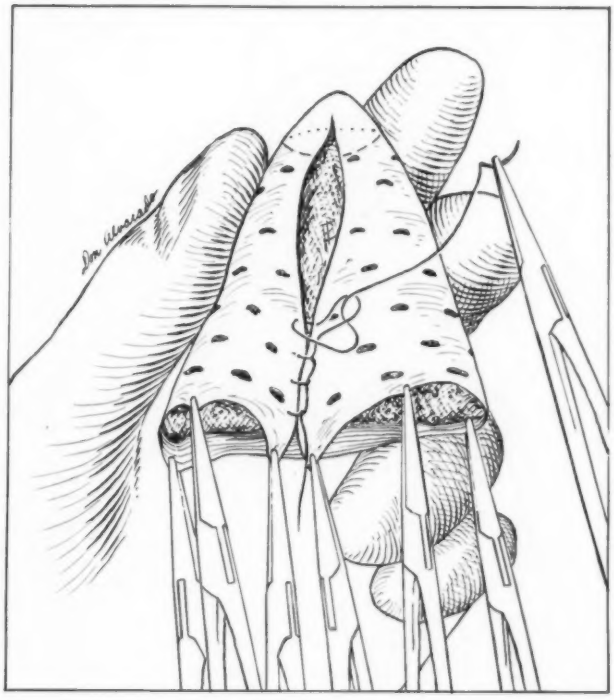

Fig. 2.—Shows the graft (dry surface outward) folded around two or three moistened 4 x 4 sponges. The resulting conical shape presents the previously described semi-circular area at its apex. This shape is maintained by a running half-hitch suture of No. 0000 plain catgut.

graft (Fig. 1) are easier to do while the skin is still attached to the dermatome drum. These stab incisions must be minute, and in that part of the graft to be applied to the middle ear no perforations are made. This is a help in assuring a sealing off of the eustachian tube orifice. Multiple small incisions seem to be more helpful when a graft is applied over bone than over soft tissue. These ear grafts are always applied to an infected area, and the perforations by providing better drainage help to prevent elevation of the graft by blood serum.

After the graft is removed from the dermatome, it is draped over the index finger or around several moistened 4 x 4 sponges (Fig. 2) in such a way that the straighter edge of the graft is folded upon

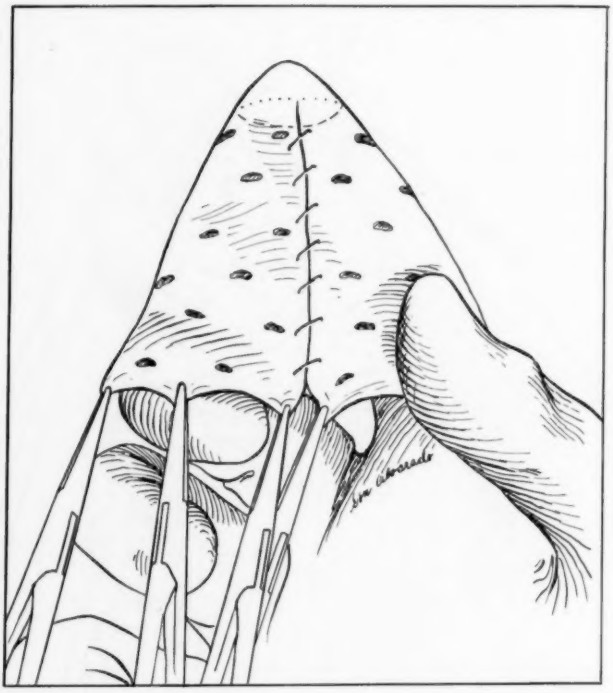

Fig. 3.—Shows the conical graft reversed so that the dry surface is now inward and the moist surface without.

itself. This gives a conical shape with the previously described imperforate area at the apex. In this stage the dry surface should be outward. A running half-hitch suture of No. 0000 plain catgut is used to approximate the graft edges and maintain the conical shape. This type of suture is used because it is faster than interrupted sutures and gives the same desired results. The graft is then inverted (Fig. 3) so that the dry surface is now inward and the moist surface without. This procedure provides a smoother seam containing minimal buried suture material. When the cavity to be grafted is a deep or narrow one and requires a conical graft of lesser breadth, this may be obtained by overlapping the approximated edges of the cone and suturing the outer edge to the surface. The conical graft is then

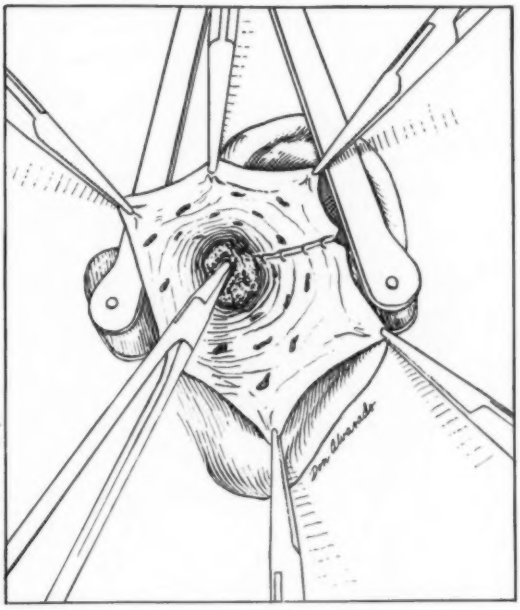

Fig. 4.—Shows the conical graft as it is set in place. A wet ball of cotton, slighter smaller than the middle ear and fixed on the end of a clamp, is placed in the apex. This fixes the graft securely as small wet cotton pledgets are packed firmly around the clamp which is then withdrawn. Moderate tension on the graft edge is maintained during the packing procedure.

In this drawing the seam of the graft can be seen running posteriorly across the facial ridge.

inverted and the redundant edge trimmed away. The graft is now ready to be applied.

Applying the Graft.—The antibiotic solution which was placed in the mastoid cavity as the graft was being prepared is now withdrawn with suction. A ball of moistened cotton, slightly smaller than the middle ear, is grasped with a straight clamp and inserted into the apex of the conical graft. With the aid of several clamps attached along the periphery of the graft to provide countertraction, the cone is placed in the mastoid cavity in such a way that the seam passes posteriorly across the facial ridge and away from the tubal orifice (Fig. 4). This seam represents the weakest part of the graft

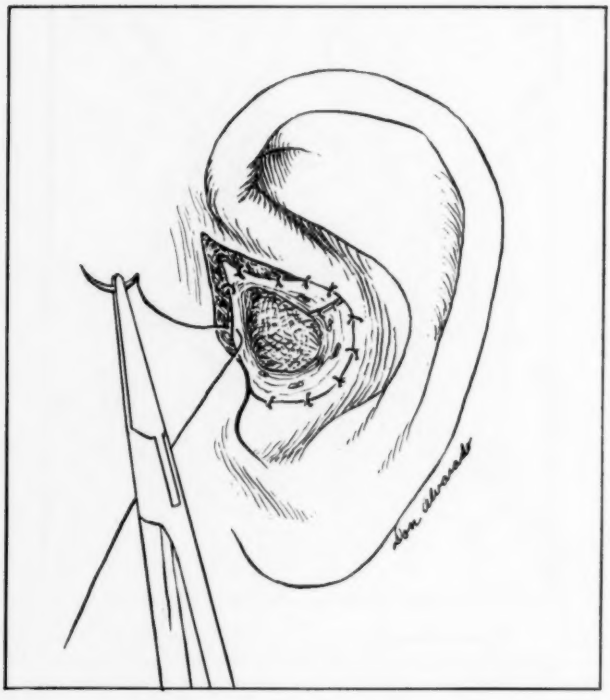

Fig. 5.—Shows the graft sutured in place with No. 0000 atraumatic plain catgut. The trimming of the graft progresses only slightly in advance of each suture. The conical graft usually overlaps the skin of the cavum but is sutured 3 to 4 mm below the skin surface superiorly in the endaural incision.

and should never follow the facial ridge, since the latter is prone to form granulations.

While the apex of the graft is held securely in the middle ear, small cotton pledgets moistened in saline are packed firmly around the clamp which is finally withdrawn. Moderate tension on the graft edge is maintained during the packing process in order to reduce inward folding to a minimum. When the graft is fairly redundant, it often facilitates the packing procedure if the periphery is clamped to the surrounding drape. This is especially helpful if the procedure is being performed without an assistant.

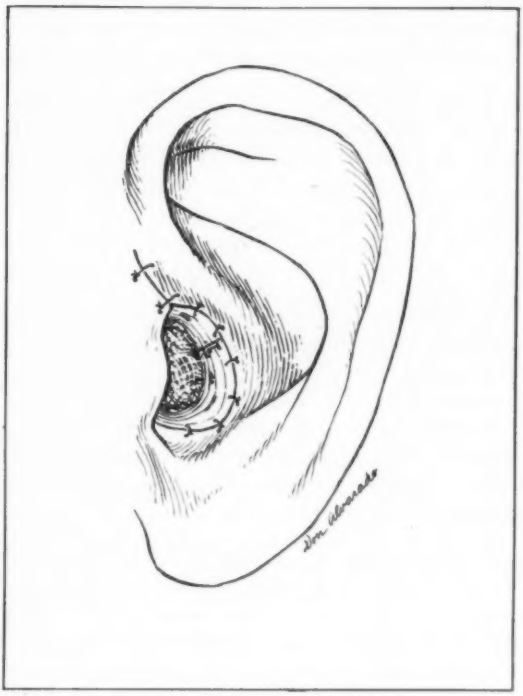

Fig. 6.—Shows the final step with approximation of the skin edges in the superior part of the endaural incision. More cotton packing is then added.

Paraffin gauze as a packing material was tried, but wet cotton seemed to conform better to the shape of the cavity and was more economical. No bad effects were noted with excessive wetting of the packing material. Small pledgets are used because they can easily be removed without causing sliding of the graft. There has been no evidence that the cotton fibers become enmeshed in the "pie crusting" but this is a good reason for keeping these perforations small.

When the cavity is partly filled with packing, the mastoid retractors are removed and the trimming of the graft begun. This trimming should progress only slightly in advance of each suture. It is wise to keep some inward pressure on the packing throughout the entire procedure. The conical graft as it is sutured in place may

slightly overlap the skin of the cavum, but superiorly in the endaural incision it should be sutured 3 to 4 mm below the skin surface (Fig. 5). Care should be taken not to suture through cartilage. Interrupted sutures of No. 0000 atraumatic plain catgut are recommended.

The final step is the approximation of the skin edges in the superior part of the endaural incision (Fig. 6). Two or three interrupted sutures of medium silk on curved cutting needles are used for this. If these skin sutures also pass through the free edges of the graft beneath, a tighter closure results. Finally, the remainder of the cavity is packed with cotton pledgets and a dry sterile dressing applied.

The After Care of the Graft.—All patients remained in the hospital for one week after surgery and all were given an appropriate antibiotic during that time.* The first mastoid dressing was on the fifth postoperative day. The cotton pledgets were removed and the cavity repacked with similar pledgets moistened with saline or dilute antibiotic solution. The dressings were always carried out with strict sterile technique. On the eighth postoperative day the mastoid area was again dressed.

The donor site dressing should not be disturbed until the eleventh postoperative day, unless it develops an odor or the patient shows an unusual temperature elevation. This donor area is usually epithelized by then although for many weeks thereafter it may exhibit a reddened appearance. If there is any bleeding from this area at the first dressing, vaseline gauze should again be applied. The patient is repeatedly warned against getting the dressing wet.

The mastoid dressing is again changed on the eleventh postoperative day and the same repacking procedure carried out. It was noted in preliminary cases that when this mild pressure packing was not maintained for twelve to fourteen days, parts of the graft would become elevated by serum and areas of slough and granulations resulted.

On the fourteenth postoperative day the packing was removed and a sterile cotton applicator used to wipe away the superficial devitalized layer of epithelial cells. This layer often comes away in large pieces and gives one the faulty impression that the graft has not taken. Also at this time any unabsorbed catgut sutures may be removed. If any granulation tissue is noted it should be cut away

*When possible the patient was given a specific antibiotic determined by preoperative culture and sensitivity studies; otherwise daily procaine penicillin was given.

with a fine wire loop. Granulations were found more often in the seam-ridge area or where the graft joined the cavum dermis. Areas of exposed dura or lateral sinus showed no tendency to produce "non-takes" or granulations. Ether is an ideal antiseptic for cleaning granulating beds as well as the grafted cavity generally. A 2 x 2 piece of gauze is kept over the external auditory canal to keep out dust and other foreign matter until the middle ear cavity is entirely healed and free of granulations.

SUMMARY

1. A method for the primary skin grafting of mastoid cavities without the use of special molds is presented. This technique utilizes a single split-thickness graft and in our series of fourteen nonselected cases the results have been very satisfactory. The entire grafting procedure requires about twenty-five minutes.

2. In each of the fourteen patients the graft "takes" were estimated at better than ninety per cent. In four, complete "takes" were obtained—primary healing. In all but one the eustachian tubes remained sealed. Of the ten cases requiring secondary healing, five were completely epithelized by the end of the fifth week, three by the end of the seventh, and two by the end of the tenth. None of the patients developed any complications.

3. A complete discussion of the technique used for all cases is given.

Gratitude is expressed to Dr. Alfred Lieberman for his interest and encouragement as well as to the other members of the Johns Hopkins Otolaryngological Staff for their advice and assistance.

848 WILDWOOD ROAD.

LIX

TUBERCULOSIS OF THE ORAL CAVITY

S. Gordon Castigliano, M.D.

and

Edward Shigeoka, M.D.

Philadelphia, Pa.

Tuberculosis of the oral cavity is an infrequently encountered lesion. It is usually seen in tuberculosis sanatoria in association with tuberculosis elsewhere in the body, nearly always as a complication of pulmonary tuberculosis. As an example of the decreasing incidence of oral tuberculosis, Oppenheim[1] in 1950, reported only 11 cases seen among 1796 tuberculous patients. Thus, today even in institutions devoting all their energies to tuberculosis, oral manifestations of the disease are rare.

Tuberculous ulcers in the oral cavity occur most frequently on the tongue, and to date over 400 cases of lingual tuberculosis have been reported.[2]

Hara and Crane recently collected 26 cases of maxillary sinus tuberculosis from the literature and reported on one case treated by them.[3]

According to Carmody,[4] the sites of the oral cavity affected by tuberculous ulcers were in the following order of frequency:

1. Tip of the tongue.
2. Border of the tongue and floor of the mouth.
3. Soft palate.
4. Anterior pillar and uvula.
5. Dorsum and base of the tongue.
6. Lower gums and jaws.
7. Upper jaw and gums.
8. Lips.
9. Hard palate.
10. Salivary glands.

From the Head and Neck Service, American Oncologic Hospital, and Temple University Dental School, Philadelphia, Pa.

Oral tuberculosis most frequently develops after the fourth decade (average age, 52.2 years) and occurs most frequently in the male.[5] This being so, the age and sex incidence are somewhat similar to that of oral cancer. Oral cancer is essentially a disease of males (80 to 85 percent). The average patient with oral cancer is about a decade older.

The typical or classical appearance of tuberculosis of the oral cavity is that of an excavating ulcer with a caseating or a granular base and an undermined edge. It is usually irregularly outlined or it may be fissured. The granular base is somewhat indurated and moderately tender when touched. By contrast oral cancer is seldom tender to the touch unless far advanced. The induration and tenderness may be varible, depending upon the stage of development of the lesion. The induration is seldom to be confused with the characteristic induration of oral epidermoid carcinoma. The initial lesion on the tongue, for example, usually develops slowly and painlessly until ulceration develops.[6] The lesion then becomes tender and painful and is associated with the passage of food or movement. Ptyalism, regarded as due to reflex secretory stimuli, may appear early. In lesions of the maxilla there may be sinus tracts communicating with the antrum.

The regional lymph nodes may become involved with a secondary tuberculous adenitis. Roentgenograms of the chest usually reveal an associated pulmonary tuberculosis. The larynx is not infrequently involved along with oral lesions. Smears made from material obtained from the ulcerated lesions by curettement and stained by the Ziehl-Nelson method show the organisms. Likewise, smears from the pharynx or sputum may be positive for acid-fast bacilli.

In a busy head and neck service in a cancer hospital such as the American Oncologic Hospital, patients with malignant lesions or lesions suspected of malignancy are funnelled from physicians and dentists covering a wide area in neighboring towns and states. Oral tuberculosis is rarely seen. In fact, because of its relative infrequency, it scarcely needs to be seriously considered in the differential diagnosis of oral lesions.

In order to stress the great importance of early diagnosis and early adequate treatment of cases of oral cancer, the Head and Neck Section of this Institution has adopted the following rigid policy: Despite the absence of physical or clinical signs of malignancy every non-healing lesion of two weeks' duration should be considered as a probable malignant tumor, and biopsy is recommended. It should be remembered that in no condition with which cancer could be con-

fused is early diagnosis of such fundamental importance to successful treatment as in cancer itself.

Although some observers[7] do not recommend biopsy of tuberculous lesions of the mouth, it must be strongly emphasized that no contra-indication exists to biopsy of an intraoral lesion. Such teaching must be abandoned. Biopsy is the only method by which an unequivocal diagnosis of cancer is established or excluded. Patients with pulmonary tuberculosis do develop cancer as do patients with oral tuberculosis. Early recognition of cancer in this group is no less important than it is in tuberculosis-free patients. Therapeutic tests of one type or another, in an effort to exclude cancer, cannot be condoned. Those early weeks are precious ones to the cancer patient. They cannot be wasted on therapeutic tests. In cancer, success depends on early decisive therapy.

During a period in which about 2200 cases of intraoral malignant lesions were seen in the Head and Neck Section of the American Oncologic Hospital, just eight cases of proved oral tuberculosis were seen, an incidence of only 0.36 percent. This is the equivalent of one case of oral tuberculosis encountered for every 270 cases of intra-oral cancer examined.

The sites of occurrence were as follows:

1. Tongue—5
   - Dorsum—2
   - Inferior aspect (bilateral)—1
   - Lateral border—1
   - Dorsum and lateral border—1
2. Upper alveolar ridge—2
3. Lower lip and buccal mucosa—1

The eight patients were all males. With the exception of one negro, the patients were all white. The average age was 52.5 years. The youngest patient was 27, and the oldest was 78. All patients had associated pulmonary tuberculosis. Diagnosis was obtained by histological means in each case. No active treatment was given for the oral lesion in four cases. These four patients were referred either to nursing homes or to tuberculosis sanatoria. Two patients (Case No. 1 and No. 2) were managed by electro-surgical excision of the local lesions followed by complete healing. One patient (Case No. 5) received 1000 mg hours of radium locally to the lesion without

benefit, and the last patient (Case No. 8) was given 25 gms of streptomycin intramuscularly within two and a half month's time with complete healing of the local lesions.

REPORT OF CASES

CASE 1.—G. W. K. (C No. 867). A 50 year old colored male patient, a bricklayer by trade, was admitted to the clinic of the American Oncologic Hospital on January 6, 1909. The chief complaint was a growth on the dorsum of the tongue of approximately six months' duration. The lesion started as a non-painful lump. The patient did not seek medical advice until about five months later, when he first saw his family physician. Treatment given was the topical application of a "liquid medicine." The lesion ulcerated a few days following the application of the local medication and became painful. The pain was aggravated by the taking of hot food.

Examination of the tongue on admission revealed a large triangular-shaped lesion, measuring 2.5 by 3.5 cms, involving the middorsum of the tongue, with the base situated posteriorly. The apex of the triangular-shaped ulcer was near the tip of the tongue. The bed of the ulcer was gray and necrotic. The ulcer edge was overhanging or undermined with a slightly indurated periphery. It was slightly tender to the touch.

There was a pleuritic friction rub, and there were signs suggestive of pulmonary tuberculosis.

The lesion on the tongue was excised by means of electro-surgery. Histopathologic diagnosis on the submitted specimen was tuberculosis.

The patient was referred to a nursing home for his pulmonary condition.

CASE 2—W. T. (C No. 3613). A 58 year old white male patient was admitted to the Head and Neck Clinic of the American Oncologic Hospital on December 28, 1938, complaining of "sores along the edge of the tongue" of approximately two years' duration. For the previous two years, the patient had been frequently distressed by multiple "painful ulcers" involving the lateral margins and tip of his tongue. About a year before admission, the patient developed a persistent cough with hemoptysis and chronic hoarseness. Anorexia with associated weight loss was noted. The patient was referred to a tuberculosis sanatorium by his family physician in April of 1937, about 16 months prior to his admission to the Oncologic Hospital.

Local examination of the oral cavity on admission revealed bilateral indurated lesions, each measuring about 1 cm in diameter, involving the infra-surface of each side of the tongue. The centers of the indurated areas were involved with multiple superficial fissure-like ulcers. No undermining was noted. The lesions were slightly tender. The regional lymph nodes were not palpable. The clinical impression was tuberculous ulceration of the tongue.

On December 28, 1938, the lesion on the right side of the tongue was completely excised, using the high-frequency cutting loop followed by electro-coagulation of the bleeding base.

The histo-pathological report on the submitted specimen was "infectious granuloma compatible with tuberculosis." The excised area completely healed within three weeks.

On January 20, 1939, the lesion on the left side of the tongue was treated in a similar manner. The specimen was diagnosed as "tuberculosis granuloma."

The patient was readmitted to a tuberculosis sanatorium, but later discharged on December 26, 1940, with the following diagnosis:

1. Far-advanced bilateral pulmonary tuberculosis with cavitation bilaterally.
2. Tuberculosis of the larynx—apparently arrested.
3. Tuberculosis of the tongue—controlled.

The patient's weight on admission to the sanatorium was 160 pounds. On discharge, it was 190 pounds. Twenty-four hours concentration studies on the sputum which were positive on admission were negative on discharge. The tongue lesions remained healed.

CASE 3.—J. F. F. (C No. 1046). A 47 year old white male patient was admitted to the Clinic at the American Oncologic Hospital on April 22, 1932, with a sore on the right side of the lower lip near the angle of the mouth of 18 months' duration. The lesion developed as a fissure following accidental self-injury by biting. About six weeks after the lesion appeared, the patient first consulted his family physician. The lesion was lightly treated by electrocoagulation. The ulcer soon recurred. About six months after the first treatment, his physician treated the lesion again—this time with the actual cautery. The lesion healed but again recurred about six months later. At that time the ulcer was painful and tender.

The patient's past history revealed "pleurisy followed by pneumonia" in 1920. Another attack of "pleurisy" occurred in 1924.

In 1928, four years prior to admission at the American Oncologic Hospital, a diagnosis of pulmonary tuberculosis was made.

On admission, oral examination disclosed a few remaining carious lower teeth. An indurated lesion presented involving the mucous membrane of the right lower lip, the labial commissure, the labial vestibule, and the buccal mucosa just posterior to the commissure. The lesion measured 2.5 cms in diameter. The lesion was centrally ulcerated. The induration in the lesion was not the characteristic induration of a malignant lesion. The submaxillary group of lymph nodes was palpable on the right side.

On April 30, 1932, a biopsy specimen was obtained from the buccal mucosa. The histo-pathological report was "granuloma compatible with tuberculosis."

The patient was referred to the Philadelphia Naval Hospital where he expired on December 21, 1937. Communication received from that institution stated that the patient's final diagnosis was tuberculosis of the lungs with involvement of the oropharynx.

CASE 4.—C. K. (C No. 3403). A 27 year old white male patient was seen in the Head and Neck Clinic of the American Oncologic Hospital on June 8, 1938, with a "sore" involving the left upper edentulous gingival ridge, of approximately one years' duration. The lesion appeared at the site of the absent first molar tooth. A biopsy of the lesion was performed by the patient's dentist two weeks prior to admission and the specimen was reported to be non-malignant.

There was a history of several attacks of pleurisy in the past ten years.

Oral examination revealed a lesion involving the left upper gum. The gingiva at the site of the upper first molar tooth was affected by a granular, slightly raised proliferative lesion, measuring 2 cm in diameter. The lesion was slightly indurated. Although ulcerated, the lesion was not tender, an unusual feature. The edges were not undermined. The regional lymph nodes were not palpable.

On June 8, 1938, a biopsy of a representative portion of the lesion was performed, under local anesthesia. The histopathological diagnosis was tuberculosis.

Roentgenograms of the chest on June 21, 1938, showed marked infiltration of both upper lobes as indicated by areas of increased density ranging from 2 mm to 1 cm in diameter.

The patient was referred to a tuberculosis sanatorium.

CASE 5.—B. H. (C No. 3151). A 65 year old white male patient was admitted to the Head and Neck Service of the American Oncologic Hospital on January 24, 1938, because of a "gum-boil" involving the gingiva in relation to the left upper first molar tooth, of about four months' duration. Because the tooth in question became quite loose, a dentist was consulted and the tooth was removed. Following the tooth extraction, the socket not only failed to heal, but increased in size, and the site of the "gum-boil" broke down and ulcerated. Two months later the dentist removed two teeth anterior to this ulcerated area. The patient was then referred to his family physician who administered local treatment without improvement. Since the lesion did not improve, the patient was referred to the American Oncologic Hospital. The referring physician's diagnosis was carcinoma of the gum.

There was a history of chronic productive cough. In addition, the patient had suffered an attack of "pleurisy" four years earlier, followed by weakness.

Examination of the oral cavity, on admission revealed a large, irregularly outlined, ulcerated lesion involving the left upper gingiva with buccal and palatal extension. It extended from the second molar site to the left canine tooth region. The ulcerated lesion involved the palate almost to the midline. Laterally the lesion extended to involve the buccal mucosa up to the level of the papilla of Stenson's duct. The over-all dimensions were 4 by 5 cms. The base of the ulcer was gray and necrotic. The periphery of the ulcer was indurated and erythematous. There was slight tenderness to touch. The left post-vascular lymph node was palpable. The clinical impression was carcinoma of the left upper alveolus.

Roentgenographic studies of the maxilla showed no osseous involvement. A biopsy of the lesion performed on January 24, 1938, showed the histo-pathological diagnosis to be necrosis, insufficient tissue for diagnosis. However, because of the clinical impression of carcinoma,* a total of 1000 mg hrs of radium was given, from January 24, 1938 to February 21, 1938, delivering 50 mg hours daily on the medial and lateral sides of the lesion. Because of the failure of the lesion to respond to this treatment, another biopsy was performed at this time. The histopathological report was tuberculosis.

Roentgenographic studies of the chest on March 7, 1938, showed uniform density involving the right base extending up to the fifth rib anteriorly. The cardiac shadow was shifted to the right. The root

*This diagnosis was not made by the present authors.

shadows were increased. Scattered over the entire right and left lung fields were numerous densities, varying from 2 to 4 mm in diameter. The roentgen diagnosis was tuberculosis. The patient was referred to a tuberculosis sanatorium.

CASE 6.—W. MCE (C No. 3982). A 46 year old white male patient was admitted to the Head and Neck Service of the American Oncologic Hospital on July 5, 1939. He had felt perfectly well until April of 1939. At that time, hoarseness and a tickling sensation of the palate became increasingly annoying.

The family physician referred the patient to a local hospital where a biopsy of the larynx was performed. No treatment was given as the biopsy was not diagnostic. In May of 1939, a month later, a "sore" appeared on the tongue. Examination at that time revealed pharyngeal and laryngeal ulcers as well. Roentgenogram of the chest showed diffuse mottling involving both upper lobes, and a diagnosis of pulmonary tuberculosis was made. In June of 1939, about one month later, another physician, who, on finding an ulcerated and indurated lesion on the tongue, referred the patient to the American Oncologic Hospital. The referring physician's diagnosis was carcinoma.

On oral examination a fissured lesion, measuring 2 x 3 cms, with a ragged overhanging edge was seen to involve the dorsal surface of the tongue anteriorly. The periphery of the lesion was erythematous and indurated. The base was gray and necrotic. Small superficial ulcers were seen in the pharynx and the larynx. The regional lymph nodes were not palpable.

A biopsy of the lesion was performed on July 5, 1939. The histopathologic diagnosis was tuberculosis. Roentgenographic studies of the chest showed pulmonary tuberculosis. The patient was referred to a tuberculosis sanatorium without treatment of the oral lesion.

CASE 7.—A. J. K. (C No. 1840). A 48 year old white male patient was admitted to the Clinic of the American Oncologic Hospital on January 18, 1915. The patient had a lesion involving the tongue, of four months' duration. Known to have tuberculosis, he had been under the care of a physician for this condition. About four months prior to admission the patient injured his tongue by biting. This was followed by a stinging sensation involving the left side of the tongue at the site of injury, with radiation of pain to the throat. A persistent ulcer developed at the site of injury.

On local examination of the oral cavity, a punched-out ulcer was found involving the left lateral border of the tongue at the

junction of the anterior and posterior tongue. The lesion measured 2 cms in diameter. The periphery of the ulcer was indurated and the lesion seemed to bear a close resemblance to carcinoma. The notable difference from the typical appearance of carcinoma was the overhanging edge the ulcer presented. Mirror examination revealed several small ulcers involving the pharynx, epiglottis and larynx.

On January 23, 1915, a biopsy of the lingual lesion showed no evidence of malignancy. Five days later, smears which were obtained were studied for tubercle bacilli. No tubercle bacilli were found. Another biopsy on this date showed tubercle formation with typical Langhans' giant cells. The histopathologic diagnosis was granuloma compatible with tuberculosis. No treatment was given and the patient was referred to a tuberculosis sanatorium.

CASE 8—T. Z. (C No. 10563). A 78 year old white male patient was admitted to the Head and Neck Clinic of the American Oncologic Hospital on January 13, 1950. One year previously, the patient had first noticed a small ulcer on the right lateral border of the tongue. The ulcer was not painful. No treatment whatsoever was given for this condition. Nonetheless, healing soon took place, without recurrence. A few months later a new "sore" developed on the left side of the tongue. This lesion was slightly painful and was aggravated by hot foods and condiments. About this time a third ulcer developed, involving the dorsum of the tongue. This lesion grew slowly in size, producing a burning sensation, at the sites of ulceration. Three weeks prior to admission, the patient first saw a physician who administered penicillin troches for the condition. The patient was then referred to the American Oncologic Hospital by his physician, under the impression that the lesion was carcinoma of the tongue.

The family history revealed that one brother had died in 1919 from "consumption." The patient is married and has three adult offsprings who are living and well.

The systemic review elicited a nocturnal, moderately productive cough. However, the patient did not complain of night sweating or hemoptysis. A history was obtained of a loss of 40 pounds in weight within the previous six months. The patient admitted that he was a heavy smoker.

On oral examination there were found two ulcerated lesions on the tongue, one on the dorsum and the other on the left lateral border. The lesion on the dorsum of the tongue measured 2.5 by 5 cms.

Fig. 1.—Case No. 8. Irregular tuberculous ulcer involving dorsal aspect of the tongue. Note flat undermined edges.

The lesion was smooth and moderately indurated, with a sharply defined overhanging edge. The base of the ulcer was grayish white in color. Some tenderness was elicited when palpated by the examining finger (Fig. 1).

A similar lesion presented on the left anterolateral border of the tongue, measuring 1.5 by 4 cms, with it greater dimension in the anteroposterior direction.

Physical examination otherwise revealed dullness of the chest in the right and left apices, with associated diminished breath sounds and moist rales. The cervical lymph nodes were not palpable. No other significant findings were noted. The tentative diagnosis was tuberculosis of the tongue, with carcinoma to be excluded by biopsy.

Laboratory examinations were negative except for smears which were made from the lingual ulcers by scraping with a scalpel. The smears were found to be positive for acid-fast bacilli. The blood Wassermann reaction was negative.

Roentgen study of the chest showed evidence of a parenchymal inflammatory disease involving the major portion of the left lung and the upper lobe on the right side. Marked areas of density were

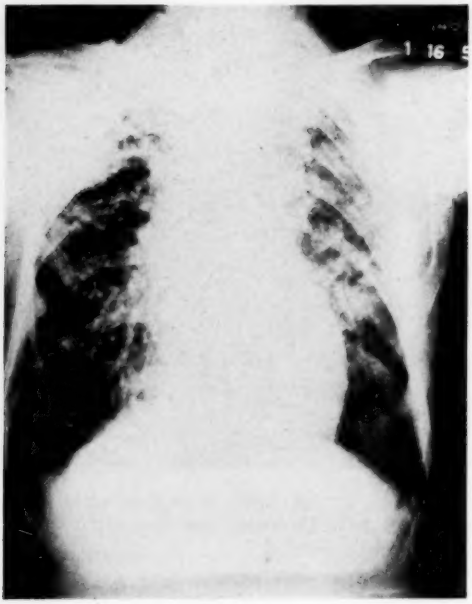

Fig. 2.—Case No. 8. Roentgenogram of chest showing parenchymal inflammatory disease suggestive of a fibroid type of tuberculosis with active disease.

noted in both apices, with a suggestion of cavitation on the right side. The changes were suggestive of a fibroid type of tuberculosis with active disease (Fig. 2). Gastro-intestinal series, including barium enema, performed on February 2, 1950, showed no evidence of disease.

Biopsy specimens taken from each ulcer were reported as infectious granuloma compatible with tuberculosis (Fig. 3).

From February 3, 1950 to April 15, 1950, the patient was placed on streptomycin therapy. One-half gram was given intramuscularly twice a day, for a total of 25 grams. There were no side effects from the use of this antibiotic. The ulcers on the tongue healed slowly and by April 12, 1950, they had healed completely (Fig. 4).

On April 21, 1950, the patient was referred to a tuberculosis sanatorium for further management of the pulmonary condition which had shown no response to streptomycin.

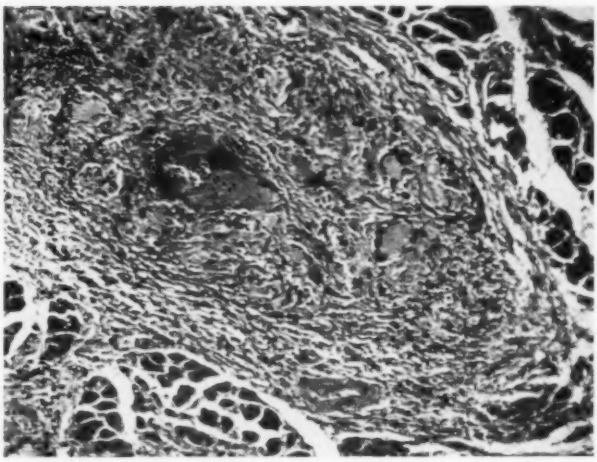

Fig. 3.—Case No. 8. Photomicrograph of tissue removed from tongue. Note the typical tubercle with giant cell formation.

SUMMARY

Oral tuberculosis is seen most frequently in tuberculosis sanatoria. But, even in such institutions, the frequency of occurrence of these ulcers is becoming relatively rare. Oral tuberculosis is so rarely encountered in the oral tumor clinic that it may be almost completely discounted in the differential diagnosis of oral malignancy. The experienced observer in a busy head and neck tumor clinic will rarely be confused by tuberculosis.

Although nearly all tuberculosis lesions of the mouth are to some extent indurated about the periphery of the ulcer, the characteristic "woodeny" induration associated with epidermoid carcinoma is lacking.

Eight cases of tuberculous ulcers of the oral cavity seen in the Head and Neck Service of the American Oncologic Hospital are reported. During the period in which these eight cases of oral tuberculosis were seen, about 2200 cases of intraoral epidermoid carcinoma were admitted for diagnosis and treatment. Each case reported occurred as a complication of pulmonary tuberculosis.

Fig. 4.—Case No. 8. Appearance of the tongue after treatment with Streptomycin. The lesion is entirely healed.

Oral tuberculosis is essentially a disease of males as is oral carcinoma. Oral tuberculosis appears in patients averaging about ten years younger (52.5 yrs.) than the average patient with oral cancer.

Nearly always palpation of a tuberculous ulcer is an uncomfortable procedure for the patient. Tenderness, then, is a characteristic feature. Where tenderness is the rule with the tuberculous ulcer it is uncommonly encountered in carcinomatous lesions of corresponding size.

Nonetheless, a tuberculous ulcer on occasion may closely simulate a carcinoma so as to confuse the unwary clinician without extensive experience in oral malignancy. However, little is lost and no harm is done unless a carcinoma is confused with a tuberculous or syphilitic ulcer and a therapeutic trial of antiobiotics is given instead of proceeding at once with a biopsy. This is the unforgiveable sin!

Unfortunately, a common error which is made in a patient known to have pulmonary tuberculosis is to assume that a persisting oral lesion is always of tuberculous origin. In some quarters, in fact, it is believed that the scientific approach in such doubtful cases is to

attempt various therapeutic tests now that quasi-specific antibiotics are available. Recent oral cancer case histories suggest such trials are on the increase.

The prognosis for a patient with an oral tuberculous ulcer is relatively poor since it generally occurs in the advanced stages of the pulmonary disease. On the other hand, it must be emphasized that an oral carcinoma in a patient with pulmonary tuberculosis may have a relatively good prognosis if the cancer is detected early and treated adequately. Delay in the treatment of oral malignancy cannot be tolerated. Those first weeks in the career of a cancerous lesion are precious ones. What is done or left undone in those early days in the course of oral malignancy may make the difference between success and failure.

Tuberculosis of the oral cavity and carcinoma of the oral cavity can co-exist. But it should be remembered that pulmonary tuberculosis associated with oral malignancy is by no means rare.

It should be pointed out that probably one in every 75 to 100 patients with active pulmonary tuberculosis past 55 years of age will probably eventually develop a carcinoma of the oral cavity on a basis of percentages. This being the case, tragic consequences will attend the adoption of any but the safe approach. The safe approach in all cases of suspected oral tuberculosis is to rule out malignancy by means of biopsy.

A biopsy should be performed on all oral ulcers or growths which do not respond to expectant treatment in a period of two weeks or less, *even though these lesions do not possess signs suspicious of malignancy in the eyes of the examiner.* Only in this way can dangerous delay be avoided. It must be emphasized that there is no contraindication to biopsy of an oral lesion, regardless of whether it is under suspicion of tuberculosis, syphilis or chronic Vincent's Disease, etc.

Streptomycin given intramuscularly is an effective method of treating tuberculous ulcers of the oral cavity. Healing of the oral lesion is noted though the pulmonary disease is essentially unmodified by the treatment. Electrosurgery may also be effective in treating some of these lesions. Tuberculous granuloma of the oral cavity is non-responsive to x-ray or gamma irradiation.

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LX

EIGHT SYNDROMES OF FACIAL PARALYSIS AND THEIR SIGNIFICANCE IN LOCATING THE LESION

KURT TSCHIASSNY, M.D.

CINCINNATI, OHIO

"Diagnosis of the approximate pathology is of utmost significance" in instances of facial paralysis, as has been emphasized by Lederer[1] in the recent edition of his monumental textbook. Recognition of the site of the lesion may provide valuable information concerning factors of etiology, prognosis and treatment.

A method of systematically examining for eight different syndromes associated with facial paralysis is described. An analysis of their functional anatomy will be made and the localizing significance of each of the syndromes will be shown.

This syndromology is restricted to lesions of those neural structures which are responsible for the facial movements per se, including the trunk from its origin in the neucleus as far down as the bifurcation at the entrance into the parotid gland, the motor nucleus of the facial nerve, and the supranuclear cortical connections. It does not particularly consider syndromes which depend on the simultaneous involvement of neighborhood structures, such as the corticospinal tract, the posterior longitudinal fasciculus and other pathways and cranial nerves such as the third, fifth, sixth, eighth, twelfth and others. These "facial symplegias," as I would like to call those associated paralyses, though of considerable significance, depend on the size and extension and/or disseminated character of the lesion. They may or may not be present in instances of identical locations of the lesions responsible for the facial paralysis.

UPPER MOTOR NEURON LESION

The first problem to be solved in any case of facial paralysis revolves around the question of whether the lesion affects the upper or the lower motor neuron.

From the Department of Otolaryngology, University of Cincinnati, College of Medicine.

The material of this paper with a few additions and changes was presented in a course given at the meeting of the Academy of Ophthalmology and Otolaryngology in Chicago, Illinois, October, 1952.

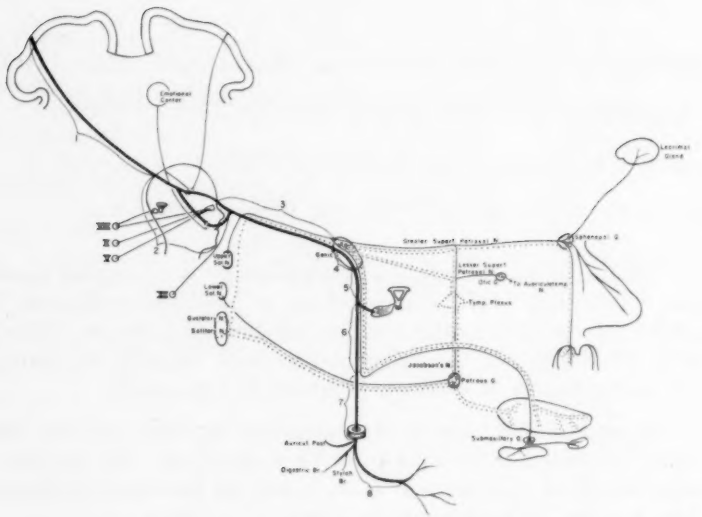

Fig. 1.—The nucleus of the right facial nerve is symbolized by the profile of a face. The heavy lines (—) indicate the crossed corticobulbar tract and the somatic motor fibers. The thin lines (—) indicate the uncrossed corticobulbar tract, the extrapyramidal connections, the anastomosis with the nucleus of the XIIth, and the secretory fibers. The dashed lines (---) indicate taste fibers and the dotted lines (. . .), fibers for common sensation.

The upper motor neuron represents pathways responsible for the transmission of impulses for voluntary movements from the cortex in the fronto-parietal region to the motor nucleus in the pons. The lower motor neuron carries the stimulus across the motor nerve to the peripheral distribution of the fibers.

A brief discussion concerning the most important neural connections of the motor nucleus and of the neural structures joining the trunk of the facial nerve beyond its nucleus might be helpful in understanding of factors significant in upper motor neuron lesion. A diagram might be useful in this regard (Fig. 1).

Supranuclear Pathways:

1. The crossed pyramidal (corticobulbar) tract. This is the connection between the cortical motor center and the motor nucleus in the pons. This pathway is responsible for transmission of impulses by which voluntary movements of the entire facial musculature on

the opposite side are initiated. The pathway and its originating nerve cells comprise the "upper motor neuron."

2. The uncrossed pyramidal tract. This is a connection between the motor nucleus and the ipsilateral cortical motor center. This connection, however, is restricted to those cell bodies in the motor nucleus which constitute the temporal branch of the facial nerve. This branch innervates only the three muscles of the forehead, namely the frontalis, the orbicularis palpebrarum, and the corrugator supercilii. Accordingly these three muscles have a bilateral cortical representation.

3. Extrapyramidal tracts. There are beside the pyramidal tracts several pathways extending to the nucleus which are responsible for the transmission of stimuli independent of conscious will. We call these pathways the extrapyramidal tracts. The most important are: (a) a connection with the presumed centers of emotion in the thalamus, corpus striatum, and substantia nigra, responsible for the transmission of stimuli activating muscles of expression by emotional impulses, and (b) a connection with trigeminal, optic, and acoustic nuclei responsible for the transmission of stimuli activating reflexly induced contraction of certain muscles. In this concern, particularly two reflexes are to be considered: (1) The blinking or palpebral reflex i.e., contraction of the orbicularis palpebrarum as a response to tactile, optic or acoustic stimuli and (2) The stapedius reflex, i.e., contraction of the stapedius muscle as a response to acoustic stimuli exclusively.

Infranuclear Connections:

Finally several pathways may be mentioned which originate in nuclei other than the somatic motor nucleus of the seventh nerve but enter into its course at various levels beyond the motor nucleus. These include:

(1) Motor fibers originating in the hypoglossal nucleus innervating the orbicularis oris muscle.

(2) Preganglionic secretory fibers originating in the upper salivatory nucleus synapsing in the sphenopalatine and in the submaxillary ganglia.

(3) Sensory fibers for common sensation and for taste originating in the geniculate ganglion and passing apparently by different pathways to the nucleus solitarius; the upper part receiving taste fibers is referred to as the nucleus gustatorius.[2]

Fig. 2.—Patient with a lesion involving the crossed corticobulbar tract, showing the weakness of the muscles at the angle of the mouth on the paralyzed side in response to the attempt to show the teeth.

Fig. 3.—Same patient as the preceding, showing normal functions of the same muscles in response to emotional stimuli.

Characteristics of an Upper Motor Neuron Lesion:

With these anatomical factors in mind, a lesion involving the crossed corticobulbar tract, the essential part of the upper motor neuron, presents the following characteristics:

1. Unilateral absence of voluntary movements of the muscles of the lower part of the face and presence of voluntary movements of the orbicularis palpebrarum, frontalis and corrugator supercilii. The patient can close the eyelids, can wrinkle the forehead and frown.

2. Absence of Bell's phenomenon.

3. Unimpaired action of all facial muscles of expression in response to emotional stimuli (Figs. 2 & 3) in contrast to what may

Fig. 4.—A typical case of Bell's palsy. Note the semifacial smiling (Janus) face in response to an emotional stimulus.

be called the "Janus-face phenomenon"* appearing in lower motor neuron lesions (Fig. 4).

4. Preservation of the blinking reflex.

5. Preservation of the stapedius reflex.

6. Unimpaired action of the orbicularis oris characterized by the ability to pronounce normally the blasting labials "b" and "p."[3]

7. Unimpaired lacrimation.

8. Unimpaired taste sensation.

9. Deviation of the chin to the normal side on opening the mouth. This phenomenon is caused by the involvement of the digas-

*Janus, a Roman deity, was pictured with a smiling and a crying face. Unilateral smiling occurs in lower motor neuron lesions and is restricted to the healthy side while the paralyzed side remains without any expression and may even show epiphora. This dropping of tears over the cheek is caused by the paralysis of Horner's muscle, a part of the orbicularis palpebrarum which under normal circumstances opens the lacrimal sac.

tric branch and therefore is restricted to lesions at or above the stylomastoid foramen, as discussed below.

Comments:

Upper motor neuron lesions as a rule are associated with hemiplegia, which is on the same side as the facial weakness when the facial neuron is damaged above the pontine decussation, contralateral when below. A lesion restricted to the site of the decussation may cause a facial diplegia of the upper motor neuron type.

I wish to emphasize that among the characteristics for an upper motor neuron lesion as listed above, only the emotionally induced mobility of the facial muscles of expression can be regarded as a diagnostic factor of absolute significance. The remainder of the signs, although regularly present in upper motor neuron lesions, may occasionally be observed in peripheral lesions if located above the entrance or below the exit of the fibers responsible for the individual function, e.g., facial paralysis restricted to the lower face may occur in peripheral lesions under such conditions as follows:

1. The lesion (tumor, injury, surgery, etc.) involving exclusively the inferior division of the facial nerve. The angle of the mouth appears to be drooping (Fig. 5) except in those cases when only the cervical branch is damaged as occurs occasionally in submaxillary surgery or radical neck dissections (Fig. 6). In these cases the angle of the mouth appears to be raised on the attempt to "show the teeth," due to the isolated paralysis of the depressor anguli oris (triangularis oris) which is an upper extension of the platysma supplied by the cervical branch.*

2. Incomplete recovery. It seems that the branches innervating the lower face, particularly around the angle of the mouth are the last ones to recover and may even remain permanently paralyzed (Fig. 7).

3. In hypoglossal nuclear lesions, the paralysis of the orbicularis oris may be misleading and suggest in addition an upper motor neuron lesion of the seventh nerve as described and illustrated in a previously reported case.[3]

*There exists also a downward extension of the platysma beyond the clavicle as far as the level of the second rib upon but independent of the major pectoralis and deltoid muscles (Piersol). This extension which provides the possibility of raising the breast independent of movements of the chest is apparently to some extent responsible for the rhythmic movements of the breast as occasionally performed by night club dancers. It is probably not too widely known that this grotesque aberration of Terpsichore depends largely on the function of the seventh cranial nerve.

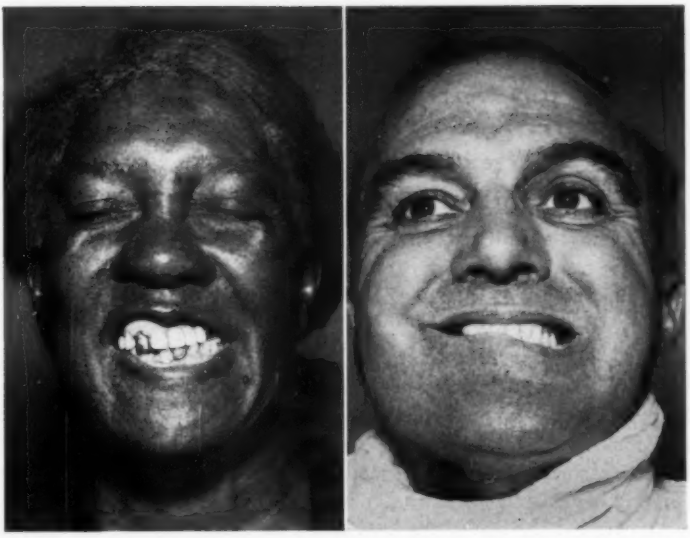

Fig. 5.—Palsy of the left lower face caused by a stab wound in the lower parotid area.

Fig. 6.—Paralysis of the right depressor anguli oris subsequent to radical neck dissection. Note the elevation of the angle of the mouth at the paralyzed side.

LOWER MOTOR NEURON LESION

The lower motor neuron is represented by the somatic motor fibers extending from their origin in the pontine nucleus to their distribution in the periphery. It forms the main portion in the trunk of the facial nerve. As mentioned before, along its course it is joined by nerve fibers which do not originate in the motor nucleus of the seventh nerve and serve functions different from those of the facial motor nerve fibers. Those nerves enter into and leave the trunk at different levels. It will be shown that the simultaneous presence or absence of all or of some of the functions provided by the branches of the facial nerve, constitute a number of different syndromes and that each of these syndromes signifies a lesion at a different level of the nerve trunk.

FUNCTIONAL ANATOMY

A description of the facial nerve in the course of the peripheral neuron is presented considering mainly factors of functional anatomy such as are relevant for this topognostic study but descriptive details are omitted. This nerve consists of four portions which are differentiated from each other by the functionally different character of their nerve fibers. Two of them are efferent (centrifugal); the somatic motor and secretory portions. Two of them are afferent (centripetal); the sensory and gustatory portions. These fibers leave and enter the common trunk at different points. This fact represents the basis for topognostic differentiation.

The Trunk:

This consists predominantly of the somatic motor portion of the facial nerve. It originates in the pontine motor nucleus, leaves the pons at the cerebellopontine angle and enters into the inner auditory meatus together with the nervus intermedius and the eighth nerve. Separating from the latter it enters into the fallopian canal. There it bends first horizontally passing through the geniculate ganglion located at the site of the knee, then vertically behind the tympanic cavity. After its exit from the skull through the stylomastoid foramen it enters into the parotid gland. There it bifurcates into an upper and lower division with several subdivisions forming finally the parotid plexus (pes anserinus) for the terminal distribution in the periphery.

Branches:

The first branch of interest for the present study is the *greater superficial petrosal nerve* which leaves the main trunk at the site of the geniculate ganglion. This branch consists mainly of preganglionic secretory fibers which originate in the upper salivatory nucleus somewhat distant from the motor nucleus. Postganglionic fibers synapsing in the sphenopalatine ganglion provide parasympathetic nerve supply for the lacrimal gland (by way of an anastomosis between the lacrimal nerve and the zygomatic nerve), for the nasal mucosa, and for the palate. It also carries sensory fibers originating in the geniculate ganglion and providing additional nerve supply for the palate. These fibers are responsible for the blisters of herpes appearing on the roof of the mouth in geniculate herpes (Ramsay Hunt's syndrome).[4] Erb,[5] in his classical scheme, assumed also the presence of motor fibers responsible for movement of the palate. He regarded paralysis of the soft palate as indicative of a lesion at or above the geniculate ganglion. His concept originally advanced by Davaine (1852) and Longet (1866) was advocated by Leube, Chvostek, Mann,

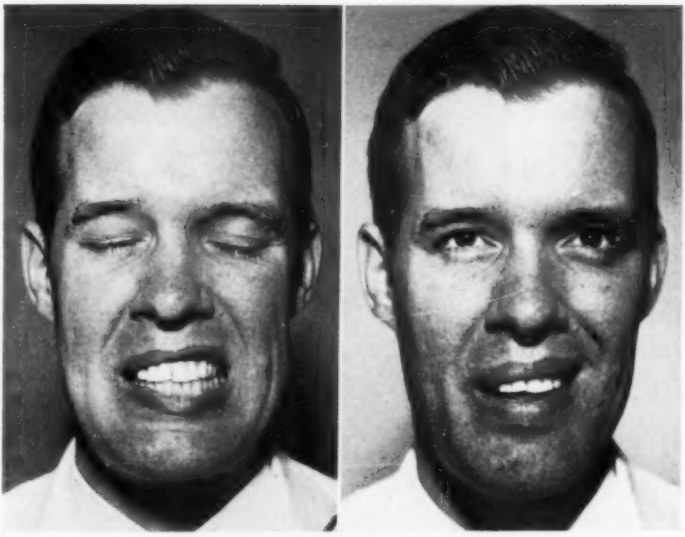

Fig. 7.—Right lower face weakness due to incomplete recovery from a Bell's palsy in childhood. Note the drooping of the angle of the mouth appearing equally on volitional and emotional stimuli.

and others, but strictly objected to by Hughlings Jackson, Gowers, Ephraim,[6] Rethi, Jacoby[7] and others and has been almost generally abandoned.[8] Most of these data are presented by Jacoby who provides a comprehensive abstract of the literature on the subject.

Next is the *stapedius nerve.* It innervates the stapedius muscle responsible for the stapedius reflex, activated by strong acoustic stimuli. Contraction of the stapedius muscle pulls the stapes out of the oval window. In this way the amplitude of the sound waves entering into the inner ear is reduced and the sound intensity attenuated. This mechanism provides protection for the inner ear. Absence of stapedius reflex manifests itself by unproportionally increased loudness (hyperacousis) and by a painful sensation (phonophobia) produced by acoustic stimuli of higher intensities.[9]

Chorda tympani. It carries three functionally different types of fibers. They are: (a) fibers responsible for taste sensation at the so-called anterior two thirds (prevallatinal portion) of the tongue, (b) fibers for common sensation at the intraoral part of the "genicu-

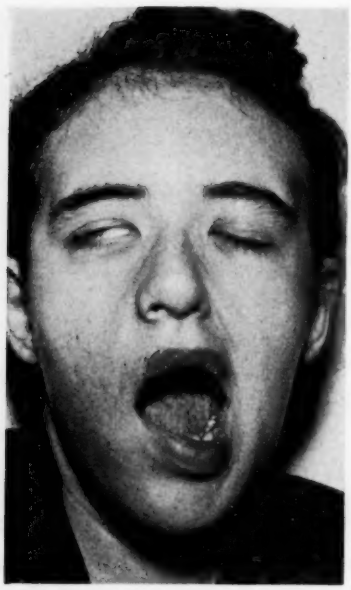

Fig. 8.—A case of Bell's palsy, showing deviation of the chin to the healthy side on opening the mouth, due to paralysis of the digastric branch.

late zone." (The presence of these fibers was postulated by Ramsay Hunt on the basis of his studies on herpes zoster oticus[4] and recently confirmed by Costen, Clare and Bishop[10]), and (c) preganglionic secretory fibers originating in the upper salivatory nucleus and synapsing in the submaxillary ganglion innervating the salivary glands at the floor of the mouth. Testing the function of these fibers reliably is almost impossible. For this reason in the present topognostic study, only the function of the taste fibers is considered. These fibers, as those for common sensation, originate in the geniculate ganglion. The latter are connected with the nucleus solitarius in the brain stem by the nervus intermedius. The course of the taste fibers however, seem to be different. Although there is some disagreement, there is reason to assume that the central connection for the taste fibers is provided predominantly by the ninth nerve via Jacobson's nerve by means of a direct anastomosis of the tympanic plexus with the geniculate ganglion or indirectly across the greater and lesser superficial petrosal nerves (Spalteholz). It is of importance to realize that the

bulk of the taste fibers leave the course of the facial nerve at the site of the geniculate ganglion. For this reason taste as a rule, is not affected in lesions located above the geniculate ganglion.[11]

Digastric branch: It leaves the main trunk below but close to the stylomastoid foramen, immediately below the posterior auricular nerve which supplies the muscles of the auricle. Erb[5] regarded paralysis of the posterior auricular nerve, manifested by the inability to move the auricle voluntarily, as significant for an intrafallopian site of the lesion. In my opinion this sign is not reliable since the majority of people can not voluntarily activate these rudimentary muscles even under normal circumstances. The digastric branch innervates the posterior belly of the digastric muscle and the stylohyoid muscle both of them responsible for opening of the mouth. It is obvious that a lesion at or above the stylomastoid foramen will necessarily cause paralysis of this branch while as a rule it will be spared when the lesion is below the foramen. In paralysis of the digastric branch the jaw deviates to the healthy side on maximum opening of the mouth (Fig. 8). Consequently the tongue appears closer to the angle of the mouth on the paralyzed side. This pseudodeviation of the tongue should not be confused with the real deviation, characteristic of a unilateral hypoglossal lesion.

Schematic Division of the Facial Nerve:

On the basis of this description the facial nerve in the course of its peripheral neuron can be divided into seven parts:

Extrafallopian (Infraforaminal). This is the most peripheral part of the nerve trunk extending from the site of its bifurcation at the entrance into the parotid gland as high up as the branching of the digastric ramus. Practically it can be regarded as identical with the part of the nerve trunk below its exit through the stylomastoid foramen.

Infrachordal. This is the part between the stylomastoid foramen and the branching of the chorda tympani.

Infrastapedial. This is the part above the chorda tympani as far as the branching of the stapedial nerve.

Suprastapedial. This is the part above the stapedial nerve as far as the geniculate ganglion.

Transgeniculate. This is the part of the nerve passing through the geniculate ganglion.

Supragenicular. This is the part located between the geniculate ganglion below and the motor nucleus above. A subdivision of this part in an intra- and extra- temporal portion may be justified, but has not yet been worked out.

Nuclear. This part is represented exclusively by the somatic motor nucleus of the seventh nerve.

Application of the Scheme for Topognosis in Lower Motor Neuron Lesions:

Lesions of the facial nerve located in any of these seven parts are characterized by different syndromes, each significant for a different level. These syndromes are based predominately on the simultaneous presence or absence of all or of some of three functions, namely, taste (in the chorda tympani area of the tongue), stapedius reflex, and lacrimation. In addition, as mentioned above, deviation of the chin to the sound side on maximum opening of the mouth and probably pseudo-deviation of the tongue can be regarded as significant of a lesion located at or above the stylomastoid foramen. Furthermore in a lesion restricted to the nucleus, no difficulty in pronouncing the blasting labials "b" and "p" will be noticed since the orbicularis oris responsible for this function is not involved.

The seven syndromes significant of lower motor neuron lesions plus the one significant of upper motor neuron lesions, as described above, are tabulated with regard to the simultaneous presence or absence of nine signs in correlation with the respective levels.

TECHNICS OF TESTING

Taste. The four cardinal taste qualities are tested by separate applications of saccharine solution, salt, quinine, and vinegar or lemon juice onto the anterior portion of the tongue with a cotton applicator. The patient is asked not to withdraw the tongue during the application of each individual test substance but to signalize the appearance of any one of those four taste sensations by raising or by dropping the right or left hand in accordance with a previous agreement, an example of which is shown in the scheme:

LEFT	↑ SALTY		BITTER ↑	RIGHT
	↓ SOUR		SWEET ↓	

Involvement of the chorda tympani manifests itself by false and/or delayed responses in comparison with those obtained from the good side.

TABLE 1.

SIGNS CHARACTERISTIC OF FACIAL NERVE LESION AT EIGHT DIFFERENT LEVELS

Level	Upper Face Movement	Bell's Phenom.	Emotional Movements	Blinking Reflex	Stapedius Reflex	Orbicul. oris Movement	Tears	Taste	Deviation of chin on mouth open
Supranuclear	yes	no	yes	yes	yes	yes	yes	yes	yes
Nuclear	no	yes	no	no	no	yes	yes	yes	yes
Supragenicular	no	yes	no	no	no	no	no	yes	yes
Transgeniculate	no	yes	no	no	no	no	no	no	yes
Suprastapedial	no	yes	no	no	no	no	yes	no	yes
Infrastapedial	no	yes	no	no	yes	no	yes	no	yes
Infrachordal	no	yes	no	no	yes	no	yes	yes	yes
Infraforaminal	no	yes	no	no	yes	no	yes	yes	no

Lacrimation. This is tested by irritating the nasal mucosa bilaterally by means of chemical substances such as kitchen ammonia or formalin solution or mechanically by means of a cotton applicator.

Stapedius reflex. In previous years, we used the strongest intensities (90 db and more) applied by means of the air receiver of an audiometer alternately to the ear on the good and on the paralyzed side. In instances of stapedius paralysis, this procedure was associated with considerable discomfort. Moreover, the danger of possibly causing acoustic trauma in an ear deprived of the protective stapedius reflex came to mind. For these reasons I restricted the use of this technic to instances with reduced hearing in the ear of the paralyzed side. Under ordinary circumstances with bilaterally equal and normal hearing, I used a technic which appears to be simple, harmless, and informative. Moreover, it dispenses with the need for an audiometer.

The "stethoscope loudness balance test" as a method of testing for recruitment. This method was originally elaborated as a test for loudness balance in instances of unilateral perceptive deafness presenting recruitment. A binaural stethoscope is placed in the patient's

ears. A tuning fork (256, 512, 1024, 2048) is mildly activated and placed in front of the bell. The patient lateralizes the sound into the better ear. The tuning fork is activated again, this time with maximal force. When recruitment is present, the patient hears the sound "all over the head" or "in back of the head" or "in both ears." During the decrement of the vibration, the sound gradually disappears at the deaf side and finally becomes again lateralized to the good ear. In the absences of recruitment, the sound is heard constantly in the better ear even when strongest intensities are applied.

The "stethoscope loudness imbalance test" as a method of testing for stapedius nerve paralysis. The same procedure can be used as a test for stapedius nerve paralysis. In this instance, of course, the sound is heard in both ears when the tuning fork is only mildly activated. When activated strongly the sound appears lateralized to the ear at the paralyzed side in the case of stapedius paralysis since at this side the attenuating effect of stapedius muscle contraction is missing.

In the case of unimpaired function of the stapedius nerve and muscle, the sound is heard equally in both ears or in the center of the head even when the strongest stimuli are applied. This test is rather sensitive. By application of the air receiver of an audiometer to the bell and gradually increasing the intensity, in instances of stapedius paralysis we have seen lateralization to ensue at intensities of 45 db.

Limitations of this method of topognosis of facial nerve lesions:

The presented method has its limitations. It can be applied with reliability only to early lesions. In lesions of longer duration partial recovery restricted to some functions may occur or by descending degeneration some functions may later be lost. Lacrimation may return very soon.[11, 12, 13] Taste sensation may come back while the stapedius reflex may still be absent. In cerebellopontine angle tumors (supragenicular lesion) taste sensation is frequently absent probably due to descending degeneration.

With these limitations in mind this method is offered as a guide for localizing the lesion in cases of facial paralysis. It represents an elaboration upon the classic scheme originally described by Wilhelm Erb.[5]

SUMMARY

Eight syndromes significant in topognosis of lesions responsible for facial paralysis are described and analyzed on the basis of functional anatomy. These syndromes are shown to differ from each

other by variations in the simultaneous presence or absence of some out of nine signs. Methods of examination are briefly described and a new simple procedure of testing for loudness balance and for stapedius paralysis is offered.

101 Louis Ave.

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LXI

THE RHINOPLASTIC MANAGEMENT OF RECENT NASAL FRACTURES

LEWIS W. JORDAN, M.D.

PORTLAND, ORE.

Recent advances by otolaryngologists in the field of rhinoplasty have brought about many improvements in the handling of acute nasal fractures. The reduction of a fractured nose can be considered as one type of a rhinoplasty and the same underlying principles apply.

All clinicians are well familiar with the patients whose nasal deformities date back to a nasal fracture which was not reduced or was inadequately handled. Many times this injury is almost totally neglected because of more major and vital injuries. Deformities of the external nose and the nasal septum may greatly disturb normal nasal physiology. Childhood nasal injuries which may appear to be minor often lead to developmental nasal and facial defects which can be difficult to correct in later years. A poor cosmetic appearance may lead to psychological disturbances. At the time of reduction of a nasal fracture, it is wise to inquire about previous fractures. The failure to obtain an adequate reduction may be due to an old deformity.

In making a diagnosis of a recent nasal fracture, the first requisite is to suspect such a condition in injury cases. Hemorrhage from the nose is usually caused by a fracture. Careful manual palpation of the nose and all the facial bones is helpful. The nasal mucosa should be thoroughly shrunken and mildly anesthetized so that intranasal palpation with an instrument is possible to find mucosal tears, septal injuries, hematomas, bony displacement, or cartilage deformities. The edema can be practically eliminated at once by the injection of hyaluronidase. In children, a general anesthetic for examination is warranted, when necessary, if the general condition permits. X-ray examination is helpful but is usually employed largely to satisfy insurance or legal requirements.

From the Department of Otolaryngology, University of Oregon Medical School.

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In times past, it was often said that early reduction could be carried out only if the swelling was slight. It is now recommended that practically all cases be treated at once if the general condition permits. Most adults can be handled under local anesthesia preceded by a mild barbiturate, and often this can be an office procedure. At times, intravenous pentothal in small amounts can be given during the actual reduction after the local anesthesia has been applied. This is best done in the hospital. Of course, all children should have an inhalation anesthesia.

In all cases, whether under local or general anesthesia, cocaine and procaine should be used in the nose. Cotton packs containing the usual cocaine and epinephrine solutions should be carefully inserted over the sphenopalatine ganglion and the nasociliary nerve for anesthesia and shrinkage. For injection, 150 units of hyaluronidase and 0.5 cc of 1:1000 solution of epinephrine hydrochloride are added to 25 cc of one percent procaine. This method is well described by Cottle.[1] Hyaluronidase is a specific enzyme which hydrolyzes the intercellular cement known as hyaluronic acid. This action greatly hastens the diffusion of tissue fluids. Because the enzyme also has the property of increasing capillary permeability, it is necessary to add a vasoconstrictor, such as epinephrine, to the solution so that it will be retained locally and not be dispersed through absorption into the blood stream. The anesthetic effect begins almost immediately and lasts the usual time with smaller amounts of solution. After its use, gentle massage easily and almost completely dissipates the edema caused by trauma and anesthetic solution (Fig. 1). In the last several months, the writer has used this solution for all local anesthesia in tonsillectomy, rhinoplasty, sinus surgery, etc. The anesthesia has been much improved and no undesirable reactions have been observed. Hyaluronidase has been widely used in surgery in recent years with practically no toxic or allergic reactions.

The injection is made on each side of the nose with a two-inch fine needle which is inserted at the alar-facial junction. It is carried up along the side of the nose as close to the bone as possible so as to anesthetize the infratrochlear and the external nasal nerves (Fig. 2). The needle can then be directed laterally and obliquely into the infraorbital foramen. Finally, it is partially withdrawn and guided horizontally under the floor of the nasal vestibule to the incisive foramen and into the columella if this is necessary. At times, injections are also made into the membranous septum and into the intercartilaginous area.

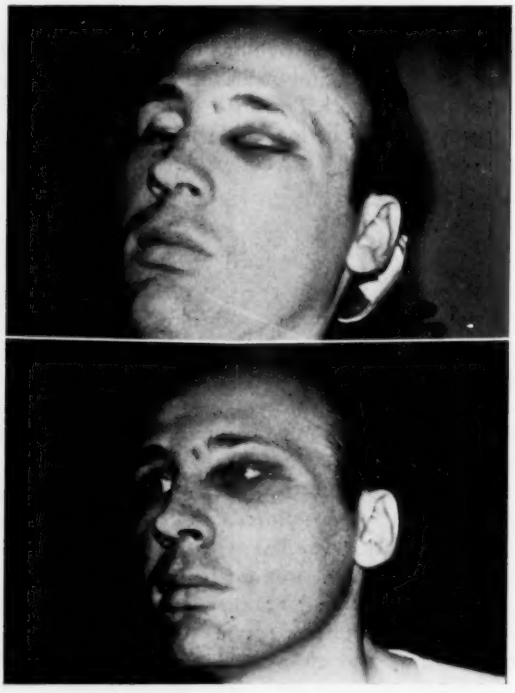

Fig. 1.—*Above*: Edema of the face 24 hours following trauma. *Below*: Decrease of edema immediately following the injection of hyaluronidase solution.

Although an unlimited variety of nasal fractures can occur, Brown[2] states that the most frequent type is that producing a lateral deviation of the entire nose. This injury may be very simple. Next in frequency is the crushing type of injury in which the bridge of the nose is depressed, and in which the extent and degree of fracture are often very great. Kazanjian and Converse[3] state that 80 percent of the fractures occur at the junction of the thick upper with the thin lower portion of the nasal bones. Often there is an overriding of the fragments of the nasal bones or of the septum. In a patient seen recently, the frontal process of the maxilla was depressed along with the corresponding nasal bone. It was necessary to elevate this entire fragment before the nose could be corrected. In another case the only injury was a horizontal fracture of the septal cartilage

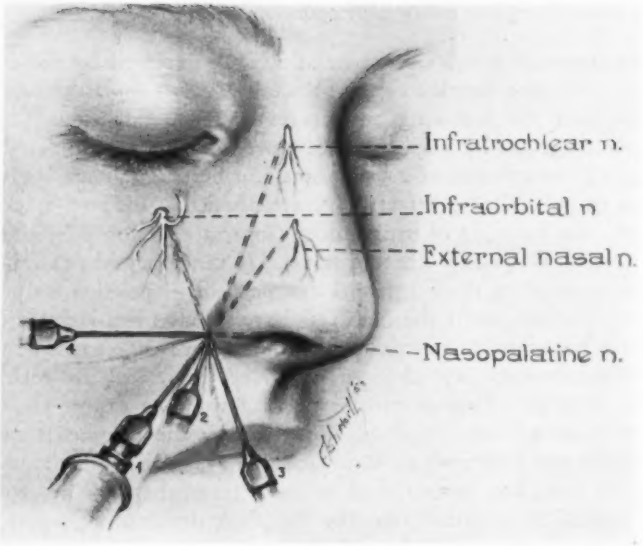

Fig. 2.—Injection of anesthetic solution.

which displaced the nasal tip laterally. Reduction could only be accomplished by the open method. Injury of the upper and lower lateral cartilages is usually associated with skin lacerations. Accurate suture is necessary to prevent later deformities caused by scar tissue contraction.

There are many simple fractures which can be easily reduced by manual pressure applied externally and a padded elevator internally. Walsham forceps are often helpful. Usually a distinct snap can be heard when the fragments are replaced. The force applied is just the reverse of that which caused the injury. Some cases cannot be reduced by this maneuver due to overriding and locking of fragments. Here, it is necessary to apply the orthopedic principles of longitudinal traction with an elevator in the nose before correcting the lateral deviation. A Kelly clamp with both blades covered with rubber can be inserted into the nose with one blade on each side of the septum for bilateral anterior traction. The fragments are molded with the fingers into the proper position.

Extreme care should be used in determining the condition of the septum. It is important to distinguish recent from old deviations. Fresh ones often are easily replaced.

In the cases which cannot be adequately replaced by the closed method, it is very simple to expose the bones by rhinoplastic incisions. Fomon[4] lists the following advantages of open reduction: (1) an accurate diagnosis by inspection, (2) precise reduction under direct vision, (3) evacuation of a hematoma and better drainage, (4) correction of pre-existing defects, (5) attention to associated fractures, and (6) the lessening of danger of postoperative deformities and impairment of function. He also states, "if the parts do not fall into easy alignment, if the fragments can be held in position only with the aid of splints, or if the cartilages have suffered considerable damage, it is better to do an open reduction." An incision can be made in the membranous septum or even farther back and the perichondrium elevated. Enough membrane is elevated to expose the fracture and to aspirate the blood clots. Often the fragments can be accurately replaced with an elevator. If not, small segments of cartilage or bone can be removed or loose fragments can be replaced into normal position between the flaps. A pre-existing septal spur can be removed or straightened at the same time, if necessary. A small absorbable suture sometimes is required. To prevent a dorsal depression in the upper lateral cartilage area, it is necessary either to have the dorsal part of the cartilaginous septum beneath the bridge intact, or to replace with cartilage or bone, cartilage in this area which was destroyed. The fragments are held in place by loose intra-nasal packing.

If open reduction of the nasal bones is required, an incision between the upper and lower lateral cartilages on one or both sides is made. The skin on the nasal dorsum is elevated as close to the cartilage and bone as possible with a knife or small scissors (Fig. 3). An excellent view of the bone is thus afforded and the pathology can be diagnosed. Reduction can usually be accomplished with an elevator or a Walsham forceps. Again, over-riding must first be corrected. Since the nasal bones and the septum are covered on each side by a membrane, they will usually remain in place if they have been adequately reduced. Suturing of either type of incision usually is not required.

Adequate packing is important in many cases. One-half inch gauze strips impregnated with cod liver oil are carefully inserted in layers with a bayonet forceps. The packing is first inserted on the floor of the nose to provide a foundation for the packing as it is

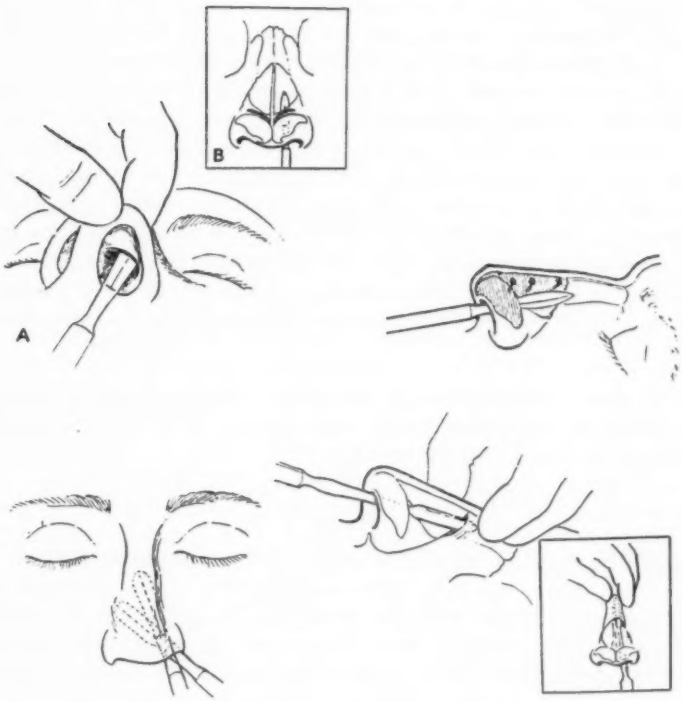

Fig. 3 A and B.—Inter-cartilaginous incision for uncovering of bony fragments; others, elevation of skin and periosteum of nose. (From Fomon)

carried up to support the septum and the nasal bones. It should not be applied too tightly because the ensuing edema may be considerable. If used in layers, it will not tend to pass down into the nasopharynx. Mucosal tears should be carefully packed in place. By means of the packing and appropriate manipulation the septum can be put into proper alignment. Further and final reduction of the nasal bones is now possible by a molding procedure using the septum and the packing as a base. This was well illustrated in a recent case of a severely comminuted and displaced nose resulting from the blow of a baseball bat. A good result was attained entirely by packing and molding. Skin lacerations should be sutured before the fractures are reduced. In the writer's experience, a headband with external fixation apparatus is rarely necessary.

Many simple fractures require no external dressing beyond a few strips of adhesive to remind the patient that he is to protect his nose. However, in fractures which are at all extensive, a firm and rigid splint should be applied to maintain the position. The nose is entirely covered with horizontal strips of one-half inch adhesive tape. Additional layers are applied over depressions. Next, two layers of thin white flannel are applied. Over this is placed a splint made of slow-hardening dental compound cut to the proper shape and held in position with adhesive strips. After the proper contour has been attained, the hardening can be hastened by ice. Adequate antibiotics can be used and the packing can be left in place as long as seven days. After a week or ten days, the large bandage can be replaced by a small lead splint which can be worn for another week.

In most cases, the results are good. The poor results usually are caused by inadequate initial reduction or tissue reaction which produces thickenings or distortions.

SUMMARY

The importance of adequate early diagnosis and treatment of nasal fractures is stressed. This is facilitated by the injection of hyaluronidase solution with the procaine. It is essential to correct over-riding of fragments before reduction is attempted. Septal fractures should be replaced. Open reduction of the septum or nasal bones may be necessary. Adequate intranasal packing and a rigid external dressing help to maintain the proper position.

624 Medical Arts Bldg.

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LXII

PHARYNGEAL PULSION DIVERTICULUM

(TRANSVERSE APPROACH)

S. L. PERZIK, M.D.

BEVERLY HILLS, CALIF.

SYNONYMS

Many synonyms for pharyngeal pulsion diverticulum are found in the medical literature. Presuming that the authors are all referring to the same lesion, these include "pharyngeal diverticulum" (Negus,[1] Dunhill,[2] Hill,[3] McClure[4]); "pharyngo-esophageal diverticulum" (Coburn,[5] McNealy,[6] Mino and Livingstone,[7] Buxton,[8] Kulvin,[9] Lahey,[10] Moersch,[11] Judd,[11] McClure[4]) "esophageal diverticulum" (Coburn,[5] King[12] Lahey,[13] Bartone,[14] Dinsmore,[15] Ross[16]); "pharyngeal pulsion diverticulum" (Shallow and Clerf[17]); "pulsion diverticulum of the hypopharynx at the pharyngo-esophageal junction" (Harrington[18]); "pulsion diverticulum of the pharyngo-esophageal junction" (Sweet[19]); "pharyngo-esophageal pouch," "pharyngeal pouch" (Graham[20]); "pulsion diverticulum of the esophagus and hypopharynx;" "hypopharyngeal diverticulum;" "retro-cricoid pharyngeal diverticulum;" "pharyngocele;" and "Zenker's terminal diverticulum." Except for the one crediting Zenker because of his classical description of the lesion, these names are all based upon the presumed location of, or the etiological factor involved in, this diverticulum.

A thorough anatomical and physiological study of this lesion points to its apparent origin in the mid-posterior hypopharynx between the oblique and transverse fibers of the inferior constrictor muscle of the pharynx. The latter transverse fibers are separately designated as the cricopharyngeus muscle. In a very interesting paper, Negus,[1] describes the comparative anatomy of this area. He shows that the assumption of the erect posture in man resulted in a straightening of the cervical spine and a recession of the mandible. This in turn crowded the now too large tongue backward forcing the epiglottis well away from its previous contact position with the

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soft palate, thus carrying the larynx caudad. Consequently two major physiological changes resulted which indirectly are related to the genesis of pharyngeal diverticula. First, there occurred an obliquity of the greater part of the superior portion of the inferior pharyngeal constrictor muscle leading to an attenuation of this muscle in the posterior midline, particularly toward its lower end. Second, due to the increased soft palate-epiglottic interspace, inspired air now reached the larynx and hypopharynx as readily through the mouth as through the nose. Since, now, man inspired air into the hypopharynx as easily as into the larynx, and since the esophageal opening, when relaxed, is many times larger than the laryngeal chink, the lower transverse fibers of the inferior constrictor muscle (cricopharyngeus) developed the function of maintaining a constant spasm in order to avoid esophageal breathing during inhalation. Furthermore, the development of independent and intensive use of the upper extremity called for a concomitant fixation of the chest wall which in turn was assisted through closure of the glottic chink. During such upper arm activity, air might tend to rush into the esophagus if it were not for the further development of the sphincteric action of the crico-pharyngeus muscle. This further accentuated the disparity between the stronger transverse (caudad) and the weaker oblique (cephalad) portions of the inferior constrictor muscle.

During the second stage of the act of swallowing, when the food bolus is propelled caudad by the progressively contracting fibers of the pharyngeal constrictors, the transverse fibers (cricopharyngeus) relax in proper time relationship to permit the passage of the mass into the esophagus. If the cricopharyngeus does not relax at the right moment, the intrapharyngeal pressure around the contained bolus is abnormally increased. This increased tension is held in check above, below and laterally by the contracting constrictors and in front by the lamina of the cricoid. The potentially weak area between the oblique and transverse fibers of the inferior constrictor muscle bears the brunt of this force which results in a herniation of the pharyngeal mucosa at this site. Frequent repetition of this phenomenon leads to a clinical herniation or diverticulum of the pulsion type. The factors responsible for the incoordination of the cricopharyngeus may be a reflex arc disturbance associated with an inadequate sensory response in the oropharyngeal mucosa, such as may be present in atrophy or scarring of the mucosa due to injuries, radiation or disease. It may be due to a spastic muscle secondary to local or distal nerve irritation. Local inflammatory changes may result in so much fibrosis of the cricopharyngeus muscle that a relative stricture is produced.

King,[12] on the other hand, feels that the original attenuation of the muscle fibers of the lower posterior aspect of the inferior constrictor is due to pressure atrophy of the muscle caught between the lower border of the cricoid and a projecting ledge on the 5th or 6th cervical vertebral body. The fact that this diverticulum is more common in the male, with his larger larynx, and has never been seen in a child with its smaller larynx and less marked bony prominences, makes him feel that this weak spot is acquired rather than congenital. On the other hand, the only well authenticated cases of posterior hypopharyngeal diverticula in infants manifested a complete muscular covering, thus pointing to their being true congenital diverticula and not pulsion diverticula which latter are hernial out-pouchings of mucosa and submucosa, with only a limited collar of thinned-out muscle fibers.

The evidence therefore, seems to point to the presence of a congenital posterior defect between the oblique and transverse fibers of the inferior constrictor muscle of the pharynx. This area remains as a potential site for hypopharyngeal mucosal herniation, which may occur in those individuals who develop a long-standing incoordination of the transverse fibers or cricopharyngeus muscle. Therefore it seems as if the name Posterior Hypopharyngeal Pulsion Hernia would be an appropriate designation for this lesion.

Many other types of pharyngeal pouches have been described. The true congenital one with the complete muscular covering has already been mentioned. Hurst described an anterior pharyngeal diverticulum which has been referred to in many subsequent publications. Morley in analyzing this case diagnosed it as a deep low vallecula and in this same article he quoted a retraction by Hurst of his original diagnoses of anterior diverticulum. Congenital lateral pouches apparently arising in the 2nd, 3rd and 4th branchial clefts have been described as emptying into the tonsillar bed, pyriform sinus and upper lateral hypopharynx. None of these are easily confused with the posterior pulsion diverticulum of the hypopharynx as it occurs in Killian's dehiscence. The pouches arising in the Lannier-Hackerman area allegedly below the cricopharyngeus and the so-called Grenz Diverticulum of Rosenthal are probably misinterpreted as to point of origin and actually represent the diverticulum under consideration in this paper.

Historically the lesion was first described by Ludlow (1764), and etiologically analyzed by Bell (1816). It was first catalogued on the basis of current appraisal by Zenker and Van Ziemssen.

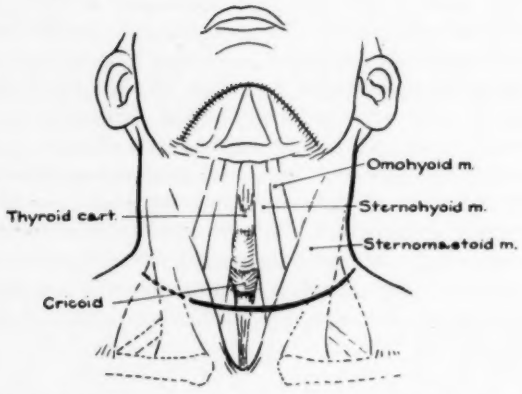


Fig. 1.—The transverse incision at the level of the cricoid cartilage with lines of extension dotted in.

(From: Surgery, Vol. 33, p. 544, April, 1953.)

SYMPTOMS

Symptomatically, dysphagia may be a very early or a very late complaint. In each instance there is a different etiological background. Early dysphagia may precede the onset of a diverticulum for months or years due to a primary marked cricopharyngeal incoordination. In the majority of cases dysphagia is complained of as a late symptom. In this group the sac is quite large and as a result of its retro-esophageal position it hangs almost directly downward with its opening in a direct line with the pharynx. Therefore food tends to enter the sac more directly rather than the anterior obliquely placed, smaller and often still spastic, esophageal opening, thus resulting in dysphagia. Regurgitation of the content of the sac, particularly with pressure on the sac after eating, is a common late complaint. The gurgling sounds on eating are due to an admixture of food and air churning in the sac. Salivation is a late symptom due to the patient's inability even to swallow saliva. In the late stages extreme weakness and emaciation appear. Due to the emptying of the sac, when the patient is relaxed as in sleep, aspiration of the content with pneumonia and lung abscess, often fatal, may follow.

DIAGNOSIS

The diagnosis is first suspected on the basis of the subjective complaints and is easily confirmed by x-ray study. A lateral film is essential in order to correlate the esophageal position with the sac.

An anterior film alone may be deceptive, especially with upper esophageal dilatation or a low-lying, deep vallecula. The antero-posterior view also notes the direction taken by the sac. The majority are reported as being on the left side, possibly because of the greater space between the esophagus and left carotid artery as compared to the right.

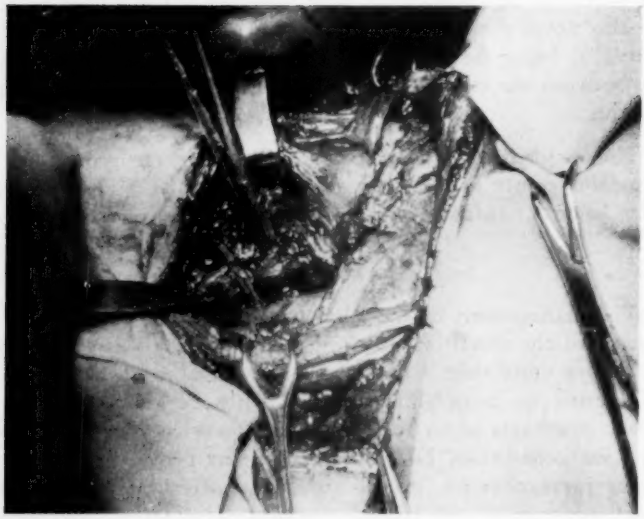
The majority of cases appear in the older age groups. There is a preponderance of almost four to one in males. The apparently similar lesions of infants are congenital and true diverticula.

MANAGEMENT

The management of these lesions is dependent upon the size of the sac and the condition of the patient. If the sac is very small, perhaps not more than a bulge or a one cm caudad enlargement, it should cause no complaints and may only be associated with the primary dysphagia of an abnormally spastic cricopharyngeus muscle. Under such conditions, bougie dilatation may prove very useful, and adjunct psychotherapy, if indicated, may often clear up or retard further progress of the lesion. Surgical attempts to obliterate the sac are often very difficult. They may result in strictures, fistulae, or recurrences more often than when dealing with larger sacs. If surgical intervention were absolutely indicated in these small lesions, invagination with plication of the surrounding muscle would offer the best chance for a successful result.

For the medium-sized sacs of 2 to 6 cm in length, surgical correction is indicated. When lesions reach this size, further stretching of the sac can be anticipated, with resulting increase in progressive symptoms as mentioned. Emaciation and the complications of aspiration can thus be avoided by performing the inevitable surgery on better conditioned patients with smaller, less inflamed sacs which have not yet entered the superior mediastinum. These lesions may be treated by several surgical procedures.

Diverticulopexy with suspension of the free unopened sac at as high a level as possible without kinking the esophagus, has been practised more frequently in the past than now. The method was devised in order to eliminate the hazard of fistula formation with resulting infection and mediastinitis, which were all too common complications of the early attempts at extirpation of the sac. The operation is relatively simple and safe but unfortunately is followed by frequent recurrences because of failure to correct the weak posterior hypopharyngeal triangle through which the still existing forces tend to produce a second or vertical pantaloon-type of diverticulum.



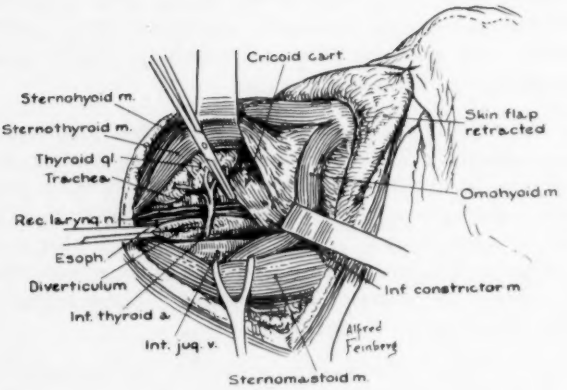


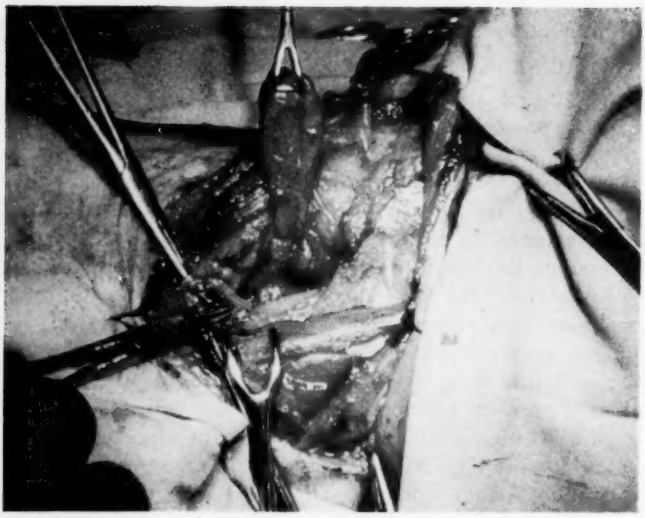
Fig. 2.—Actual and diagrammatic surgical field revealing the extent of both the transverse and vertical exposure through the collar-type incision; the diverticulum lies in plain view posterior to the esophagus and deep to the inferior thyroid artery.

(From: Surgery, Vol. 33, p. 544, April, 1953.)

Not infrequently the suspended sac relaxes from its position and become sufficiently dependent so that refilling with food recurs. If the sutures are put too deeply through the apex of the sac, infection may follow.

Inversion of the sac with two layer repair of the defective pharyngeal triangle gives promise of being a safe and successful alternate method of treatment. Here again the operation has the double virtue of being performed easily and on an unopened sac. The drawback of recurrence as experienced with diverticulopexy is practically eliminated in this operation since a double layer plication of the surrounding constrictor muscle fibers is performed subsequent to the inversion of the sac. It is reported by those having the greatest experience with this procedure that the sac tends to shrink almost completely within six to eight months. Whether it can be performed with the very large sacs without producing esophageal obstruction is open to question. The possibility of accidently opening the neck of the sac while teasing it off of the attached musculature might result in a non-detected leak with serious infection. As yet there are not enough reports at hand regarding the recurrence rate but the procedure has been performed often enough with success by many surgeons to warrant its serious consideration in a selected group of cases.

Excision of the sac in one or two stages is currently the most popular method of surgical management. When first performed the one-stage procedure was followed by severe complications and sequelae. These were local contamination, frequent mediastinitis and abscess and fistula formation due especially to the lack of our modern methods of controlling infection. In addition, as pointed out by Sweet,[19] the management of the pharyngeal opening with crushing clamps, thermal cautery, or carbolization and the use of heavy catgut with inversion of the purse-string ligated stump into the wall of the gut, were often responsible for these complications. At times a drain in contact with the suture line would further assist in the initiation of this destructive process. On the other hand an absence of external drainage with the formation of a blind internal fistula could result in local infection dissecting down the prevertebral space toward the mediastinum. Under such circumstances it is easy to see how the introduction of the two-stage excision of the sac was avidly accepted by surgeons. Although these same infectious complications occurred they were less frequent and not associated so often with mediastinitis. It was felt, however, that a better controlled one-stage procedure would lessen the period of hospitalization, the hazard of two anesthetics and the frequency of recurrences. With



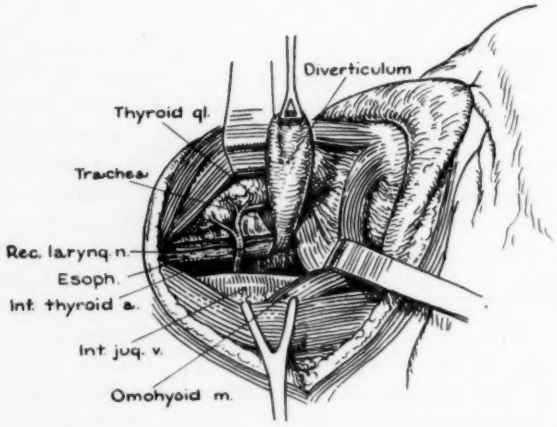


Fig. 3.—Demonstrating the satisfactory exposure of the surgical field during the freeing of the diverticulum; note the limited manual retraction required; the recurrent laryngeal nerve is in full view and separated from the neck of the diverticulum by the full thickness of the esophagus.

(From: Surgery, Vol. 33, p. 544, April, 1953.)

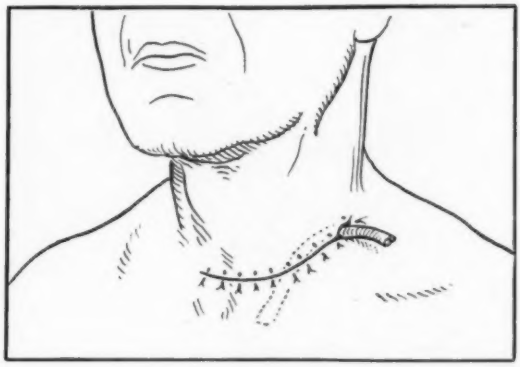


Fig. 4.—Closure showing the drain directed toward the superior mediastinum and away from the pharyngo-esophageal suture line.
(From: Surgery, Vol. 33, p. 544, April, 1953.)

the advent of more meticulous management of the pharyngostome and the use of anti-infectious agents, it is easy to understand why the one-stage excision of the diverticulum has become the procedure of choice.

ONE-STAGE TRANSVERSE APPROACH

With very few exceptions the transverse collar incision (see Figs. 1 to 5) as used in thyroid surgery offers the best exposure and cosmetic result. This approach allows for the same up and down delineation of the surgical field as is accomplished with the vertical incision and in addition gives a much increased expanse in the transverse diameter. As a result, the sternocleidomastoid muscle can more easily be retracted laterally and the neck viscera can more readily be rotated medially on their vertical axis to bring the neck of the sac into the unobstructed center of the surgical field. Likewise if it becomes necessary to explore the opposite side or deal with a bilateral or transverse pantaloon type of sac, this can be done without the necessity of making a second vertical incision on the other side of the neck. The transverse incision is about 3 cm above the clavicle and extends from a point about 3 cm lateral to the posterior border of the sternocleidomastoid muscle to terminate 3 cm to the opposite side of the midline. The incision can be placed primarily on the side of the neck selected, and can then be extended in either direction as desired. The anterior border and under surface

Fig. 5.—Two months post-operative; satisfactory cosmetic result.
(From: Surgery, Vol. 33, p. 544, April, 1953.)

of the sternocleidomastoid muscle must be freed from the level of the hyoid bone down to the suprasternal notch. While retracting this muscle laterally the anterior belly of the omohyoid muscle is exposed. This muscle may be transected or retracted either superiorly or inferiorly as indicated by its position and as determined by the length and breadth of the neck. Usually it is transected or retracted superiorly. The internal jugular vein, carotid artery and vagus nerve are now exposed and gently retracted laterally. This maneuver cannot be fully accomplished until the middle thyroid vein is transected. At times, the inferior thyroid artery lies in firm contact with the sac so that it must be transected. At this point great care must be exercised not to injure the recurrent laryngeal nerve for the latter runs either deep between or superficial to the main trunk or the first branches of the inferior thyroid artery. Before the artery is cut, the nerve should be located, usually where it dips deep to the lower margin of the inferior constrictor muscle at the lower level of the palpable cricoid cartilage, almost parallel with the posterior tracheal border. This is a relatively fixed point. With the recurrent laryngeal nerve in full view, it cannot be injured. It is now possible to sharply rotate the larynx and thyroid to the opposite side although in a very few cases it may be necessary to even transect and ligate the superior thyroid vessels. If the diverticulum cannot be immediately visualized as it lies posterior to the esophagus as a sort of re-

duplication, palpation of the inferior border of the cricoid will immediately and always indicate the position of the neck of the sac. The diverticulum must originate at this point posterior to the esophagus and extend caudad for a variable distance. The enveloping esophageal fascia is now opened and the sac is freed. As its tip is picked up with an instrument, it is carefully dissected until the neck is exposed. The latter is usually wider than is apparent because a lateral displacement gives the neck a narrow and one-sided appearance By sharply rotating the head, larynx and thyroid, the neck of the sac can be readily visualized in its full transverse position. The muscle fibers covering the first one to two centimeters of the neck are cleared and carefully preserved as a cuff, exposing the submucosa. A temporary stay suture is placed at each end of the neck of the sac. At this point one must be careful not to take too much of the main gut or leave too much of the neck of the sac. Short bites are then taken on the neck with a straight mosquito clamp and the neck is progressively incised step by step on the proximal (esophageal) side of the clamp. Four (0000) or Five (00000) interrupted silk sutures are then placed in the non-traumatized mucosal edge and gently tied with the knots on the inside. A second similar row of sutures approximate the previously developed muscular cuff and the gut is returned to its prevertebral position. It is not necessary or desirable to suture the gut to the prevertebral fascia. All the previously exposed organs, vessels and muscles are permitted to fall back into position without suturing. The skin margins including the platysma are approximated as in thyroid surgery. A small penrose drain may be barely tucked through the lateral end of the wound, but well away from the pharyngeal suture line.

We have never found it necessary to use an esophagoscope to guide us to the sac during its dissection nor have we found it necessary to pack the sac with gauze.

Although the one-stage excision warrants first choice in the surgical management of a pharyngeal pulsion diverticulum, there are some cases that lend themselves more safely to a two-stage procedure. This is the group of very large mediastinal sacs with much inflammation in the elderly, debilitated and starved patient as described by Mino and Livingstone.[7] In these patients it is often necessary to perform a preliminary gastrostomy.

POSTOPERATIVE CARE

Feeding tubes are not used. Oral liquid diet is begun when the patient has fully reacted. This is changed to soft food on about

the fifth day. Normal feedings are instituted about the tenth to the fourteenth post-operative day.

The drain is removed on the first or second post-operative day and all of the sutures by the second day.

Antibiotics are given empirically and prophylactically beginning twenty-four hours before surgery.

The patients are out of bed when fully reacted and usually leave the hospital by the fifth to the tenth day.

CONCLUSION AND SUMMARY

Various theories regarding the etiology of pharyngeal pulsion diverticula are reviewed. The lesion, as usually seen, is an acquired herniation of pharyngeal mucosa through a congenital defect in the inferior constrictor muscle of the pharynx.

The management is simplified by the elimination of the guiding esophagoscope during surgery and the feeding tube post-operatively.

The transverse incision for the surgical repair of this lesion is presented as a more efficient and cosmetically acceptable approach.

300 S. Beverly Drive.

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The Scientific Papers of the American Otological Society

LXIII

ADDRESS OF PRESIDENT

ALBERT C. FURSTENBERG, M.D.

ANN ARBOR, MICH.

Members of the American Otological Society, and guests:

Each year it is the duty of the President on this occasion to address the members and guests of the American Otological Society. The President has no escape from this responsibility. He is adjured by the precedent and tradition of eighty-five annual scientific meetings to respond in his very best manner at this time. He may have tried to delegate this part of the program to other more distinguished members of the Society but with no success. On each occasion he is reminded magnanimously of the superior privileges of the President and cajoled to exercise his prerogative at this important funtcion. One distinguished member of the Society, frank and less felicitous, asked me to spare him from a grave and expounding discourse but nevertheless to discharge the duties avowedly those of the office of the President no matter how disagreeable or reprehensible they might be.

Again, precedent and tradition have prescribed more or less what the President is supposed to say on this occasion. He is expected to disport himself intellectually with a scientific discourse in otology or with philosophical advice and admonitions to the members of this group. However, I am going to veer away from the customary gentle reproofs and kindly warnings of a President and talk to you briefly about some of the present day problems of medical education. I feel certain that I am in the midst of a receptive audience. Many of you are faculty members of a medical school. A goodly number are fathers of medical students or the parents of prospective candi-

dates for the study of medicine. All of you are interested in the purposes for which a medical school is maintained. I shall not urge or enjoin you to make commitments, acquire any prejudice, or take action at this meeting. I merely desire to call your attention to certain problems of medical pedagogy which demand solution, which will be solved, and in which you, as practitioners of medicine, are as vitally concerned as any of your colleagues who happen to hold positions in an institution of higher learning.

You will agree, I am sure, that our first purpose in medicine is to acquire that quality of training which will make it possible to practice our profession with credit to ourselves and safety to our patients. In this connection, it is the solemn obligation of our medical schools to select good timber. They must choose students who are properly prepared to study medicine. The need of a broad general education for the demanding disciplines of medicine was recognized many years ago. One's responsibilities in our profession reach beyond those of solving mere technical problems; they demand much larger attainments, a civic and social consciousness, the ability and desire to contribute to the progress of our profession and the acquisition of those cultural and moral values which promote human welfare and humanitarian efficiency.

The great men of our profession are not those who have assigned themselves to specific technical jobs on fixed schedules of working hours. The men of prestige and leadership of national scope in our field are the strong, resourceful individuals who have developed comprehensive and effective programs of instruction, research, or service, which redound to the benefit of mankind.

It is of interest to recall that after World War I the medical schools of this country were deluged by applicants for the study of medicine. The same situation prevailed after World War II. In the years 1947-1950, three times as many qualified students as could be accommodated pled for an opportunity to study medicine, while since this period and notably in the past two years, many of the 79 medical schools of this country have had difficulty in enrolling entering classes of high scholastic attainments. For reasons not easily explained, we now observe a definite decrease in the number of well qualified students seeking admission to the study of medicine. As a matter of fact, many of the medical schools this year will scrape the bottom of the barrel for their entering class next autumn.

But one can predict with strong convictions that this condition is of short duration. A note of warning must be sounded to those institutions which are content with the status quo and serenely satisfied with the funds and facilities now at their disposal. These colleges and universities seem destined for a rude awakening. If one estimates the number of students in elementary schools today between the ages of 9 and 11, and is willing to accept the premise that the young people of the future will be as interested in higher education as those of the past, our colleges and universities must anticipate a 50% to 75% increase in enrollment by 1960. The percentages are even higher when one considers the probability of an ever increasing demand for the baccalaureate and professional degrees in the years to come.

To cite an example, I might say that there are enrolled at the University of Michigan this year approximately 17,000 students. The administrative officers of the University are giving serious consideration to plans for the admission of some 28,000 to 30,000 in 1960. When and if this occurs, the impact of this clamor for higher education is certain to be felt by our schools and colleges of medicine. Privately endowed and state supported schools will not be immune to the pressure of alumni and legislators, and the demands of public opinion. When the time comes that many more well qualified young men and women knock on the doors of the medical schools than can be accommodated, these pressures quite properly become a potent influence on the administrative and fiscal policies of any institution. And this is all the more aggravated by the fact that there is a need today for more physicians in this country. The fields of psychiatry, public health, roentgenology, pathology, pharmacology, medical research, and academic medicine in all the basic sciences are so barren that there seems little chance of bringing them up to par without prompt and drastic increases in enrollements of our medical schools. In an effort to discharge our obligation to this public need we at Michigan have admitted entering classes of 200 students since 1951, a 33 1/3 percent increase over previous years.

The keenest imaginations of medical men today are challenged by the all important problem of adequate medical service for all groups of our population, economic as well as geographic. We come face to face daily with that all important controversial issue which deals with the number of physicians in this country. We hear from

all quarters that there are too few doctors in the United States; that we are not training them fast enough to keep pace with loss by death and retirement and that this situation will become more and more critical each year unless steps are taken to furnish instruction to a greater number of medical students. Some of the accusations are unjust; nevertheless, all of them are important. There is a challenge before us today to bring together some sincere, honest, and intelligent men from the ranks of medical educators, administrators, practitioners of medicine, and the public to investigate the fundamentals of medical service and to develop, if possible, a unity of thought and agreement on this issue. To formulate a policy we need an honest, factual, non-emotional evaluation of the requirements of adequate medical care and a practical program of action.

Statistics for various purposes and design are presented to us in great abundance by many persons with ethical differences of opinion. One individual, for example, tells us that we have one doctor for every 749 persons in this country and thus we are adequately supplied with medical service. Another informs us that while these ratios may be true now, we are, nevertheless, facing a period of decadence because of our failure to reckon with anticipated normal mortality losses and attrition of medical men, military requirements, increasing public demands for service, and growth of population. I question the validity of all these figures. Frankly, they have me confused. They have succeeded in doing what Stephen Leacock said could be accomplished by any academician addicted to numeration. "If, when delivering a statistical speech," said Leacock, "you observe that some member of your audience is awake and appears to have a gleam of intelligence on his face, throw in a fraction or two, and you will promptly put him to sleep." That is precisely what has happened to many of us. Speakers on this subject have thrown too many fractions at us. I am no doctor of economics but it is obvious to me, and it must be to you too, that we need more doctors if you can't find one when you are in need of help. The law of supply and demand is fundamental and easily understood by everyone. If a doctor is not available when his services are required and appointments must be made sixty to ninety days in advance, surely there is something wrong with the supply of medical talent. I, for one, believe that there are valid arguments for increasing our present enrollment in the medical schools of this country and that if this is done in a judicious and orderly manner, we need have no apprehension about ful-

filling public demands for medical care and at the same time maintaining high standards of medical education. Where human lives are at stake, there can be no compromise with that fundamental principle of the highest possible quality of medical service for all groups of our population.

A greater crisis perhaps involves the distribution of medical care and here a great public responsibility must be recognized. The doctor strives for a successful career in his profession. Success for him is, in a very large measure, evaluated by his ability to practice medicine in a creditable manner and to render a quality of service comparable to that which he has observed during his clinical years in the study of medicine. He seeks recognition in the medical profession; the admiration and esteem of his fellow doctors, and he knows full well that these attributes are gained not alone by dint of effort but also by the availability of modern facilities for the practice of his profession. Adequate and satisfactory distribution of medical service demands that the practice of medicine be made more attractive in small communities if physicians are to be lured away from large metropolitan areas. Occasionally the young doctor who is financially encumbered commences the practice of medicine in a small village but strives to abandon it when his finances will permit. Astonished residents in a dilemma, cannot understand why the doctor with such a thriving practice departs from their community. To join them is to remain forever is their axiom. Obviously, the magistrates and elders of these villages are imbued with the same complaisance that prompted someone to place a sign I saw the other day over the gate of a cemetery in Dearborn, Michigan which reads "Entrance Only."

The progress of medicine in this country during the past two decades has been phenomenal. I need not take your time to dwell on the colossal achievements of research, their availability in public health, and the great advances in medical service which America is now enjoying. Moreover, promise lies most hopefully in the discoveries which are destined to be made soon in the degenerative diseases, the major causes of disability and death today.

However, medical education now is at the brink of disaster. Most of the 79 medical schools which have contributed so significantly to the high standards of health in this country have come face

to face with serious financial problems which may wreck the foundation of medicine culminate in a national disaster.

The seriousness of this situation cannot be exaggerated. The Commission on the health needs of the nation has estimated that the number of doctors required by 1960 will vary from 22,000 to 45,000 more than the anticipated supply. Yet, the income of medical schools today is notoriously insufficient to meet the costs of operation. Threatening financial deficits are making it exceedingly difficult for many of the medical schools to maintain satisfactory teaching standards. Competent teachers for the basic sciences are at a premium. These fields are exceedingly barren because of the great demand for the services of physiologists, biochemists, pharmacologists and pathologists in industry and private hospitals. Many of our highly qualified academicians have been lured away to positions in these fields which offer salaries of two to three times those provided by a medical school budget. The net result is obvious. Faculty time for the individual medical student has decreased 7% in the past decade. I wonder if many of us realize that the salaries of men and women in academic fields are actually 10% less than they were in 1940. Well the students on our campuses know this and are shying away from the teaching profession.

The doctor of today is interested in his livelihood; in making a satisfactory living for himself and family. I am unable to reconcile to modern medical practice the time worn tradition that the doctor enjoys starving in his attic. In spite of idealistic pronouncements to the contrary, it would seem to me that a man virile enough to be a good doctor is also sufficiently hearty to enjoy his food. Those sensitive souls who talk about one's love of science for science sake and believe that those engaged in it do not claim any utilitarian value for what they accomplish, have never had the administrative experience of a Dean. The great men of medicine who have created epochs in the progress of science did not starve in laboratories. At least the court proceedings of their probated estates do not reveal many instances of such a tragic ending.

The cost of training a doctor is exceedingly high today. Most medical schools contemplate an outlay of $10,000 to $12,000 to train a doctor of medicine. Some institutions anticipate much larger sums in the immediate future. Teaching budgets have increased 94%; administration 116%; plant operation 45% in the past ten years.

Tuition fees which have risen 165% since 1940 still provide only 1/5 of these costs.

It is estimated that the 79 medical schools of this country operated at a deficit last year of ten million dollars. The income from endowments, foundation grants, private gifts, and general university funds are insufficient to meet current operating budgets, not to mention the future requirements of most schools. Unless support is forthcoming from some source, private, state or federal, many medical schools will be forced to lower their standards of training or close their doors entirely.

We recognize three important responsibilities in a medical school, namely, medical education, care of the sick, and medical research. There is no line of demarcation between these three functions. Each is inexorably dependent upon the other two. A medical school is only as strong as the chain that links together in intimate relationship good teaching, sound research, and unimpeachable practice.

Where there is no reesarch there can be no progress in medicine. The outstanding doctors of today are strong, deliberative persons who are making contributions to our knowledge of medicine. They are imaginative men and women who derive theories from their observations and strive by careful experimental work to reduce these theories to facts. Those doctors who promptly become aware of these facts, put them to practical usage and thereby extend the boundaries of humanitarian service in the prevention and treatment of disease likewise are important contributors to medicine.

Creativeness is the greatest thing in life. What a joy and satisfaction it is to discover some useful treatment or to develop a surgical technique that changes the failures of the past into the successes of the future, and what an enviable position we hold in our profession. A wealth of fundamental facts are available to us today; scientific precepts which have laid sound foundations for investigation upon which we may build superstructures of inestimable value in the prevention of suffering and the saving of lives.

In spite of some pessimistic pronouncements which I have addressed to this Society, I should like to say without equivocation that there need be no apprehension about the future of otolaryngology if we will maintain adequate teaching programs, obtain the best training that is available and improve the quality of our services

from time to time through the utilization of post-graduate opportunities for continuing study. We shall not be required to practice otolaryngology in any form that will sacrifice the best interests of our profession if we remain alive to the changing needs of our social structure and assume the office of a professional guide to whom the public delegates with confidence the duty of advising society how changes should be made. Our greatest security lies in our own inherent ability to bring about rational and orderly changes from within our own ranks. Let us hope that we will not fail to discharge this solemn obligation to society and our profession.

University Hospital.

LXIV

OTOLOGY IN PERSPECTIVE

H. I. Lillie, M.D.

Rochester, Minn.

Mr. President, Members of the Society and Guests: It is with sincere gratitude that I express my deep appreciation of the honor your president, Dr. Furstenberg, has bestowed on me by inviting me to be his guest of honor at this annual meeting of the American Otological Society. It is difficult to reduce to words the emotion that is present in my heart. I had had an extended vacation during the cold months and had not thought that I would attend the annual meetings, but this opportunity could not be declined.

The president needs no eulogy for his outstanding attainments in the medical and teaching professions. As dean, his great administrative ability and foresight have made the medical school outstanding.

As an author, he has contributed many erudite, basically sound and authentically scientific articles which have added greatly to our knowledge. These contributions will stand the test of time. He has been an actively contributing member of the special societies for a number of years. His presence on programs has added greatly to the attractiveness of medical meetings. In recognition of his achievements in the profession, medical honors, justly deserved, have been bestowed upon him. His career has been a full one. How he finds time to do all the things he does is beyond my comprehension.

Many of you may not know it, but I feel as though I had something to do with his choice of otolaryngology as a career. When he was an undergraduate student I urged him to accept an appointment for training with Dr. R. B. Canfield. He has never left the University of Michigan. I am proud that I may have had something to do with his distinguished career, as you can well appreciate.

Since my entry into the practice of otology, the specialty has made great progress in clinical aspects and has made greater progress

in scientific aspects of the intricate problems that present themselves. The welfare of the patient has been enhanced to a degree scarcely conceivable forty odd years ago.

To enumerate a few important advances, I think one would consider first the great improvement in graduate teaching and training in the specialty. There were few centers where adequate training in this country was available forty odd years ago. Those that were available could accommodate all too few of the serious-minded aspirants. Many of those desirous of training availed themselves, at great sacrifice, of the organized training carried on in Europe, especially in Germany. In Boston, New York, Philadelphia and Ann Arbor, training services fashioned after the best in Europe were organized. At the present time there are available in this country more centers for training than are available in Europe. In the early days, if one was a deft surgeon he was considered to be a good otologist. Not now, however, because at present to be a good otologist one must be a good physician, skilled in accurate diagnosis, familiar with many basic scientific facts concerned with the cause of symptom complexes, and able to utilize to the advantage of the patient the remedial agents available for rational treatment.

Improvement in the clinical and surgical aspects of the suppurative diseases of the temporal bone would be second in importance it seems to me, as early recognition of serious suppurative disease of the temporal bone was formerly not the rule to any great extent. When signs of subperiosteal abscess developed, it was deemed advisable to perform an emergency mastoid operation, not with the completeness as of today, but merely to the extent of opening the cells as a rule, thus permitting drainage to take place. The use of open packing of the wound was in vogue. This resulted in great deformity and long convalescence. Actually the mastoid operation was feared by patients and physicians alike. It was no wonder that so many cases of chronic suppurative otitis media and mastoiditis resulted, with the morbidity and mortality that we now know could have been avoided.

Gradually courageous younger men began to think for themselves and, with considerable misgiving, to suggest improvement in diagnostic criteria and technical procedures in medical society meetings such as this. The programs were comprised of subjects dealing with the clinical and surgical aspects of suppuration of the temporal bone, for the most part. Free discussions were the rule.

The adjunctive information obtained from roentgenograms was not fully appreciated or understood. Roentgenographic findings have gradually become extremely important as evidence of disease of the temporal bone. Cytologic examination of the blood was available and was understood but the intricate chemical examinations of the blood so generally used at the present time were not. Knowledge of the pathologic processes of the temporal bone was gradually increased until at present these processes are fairly well understood. Actually the rational treatment of disease of the temporal bone was improved to the point that surgical procedures were no longer feared to the extent they formerly were.

The advent of supportive transfusions of blood when indicated and the adequate use of specific therapeutic agents such as sulfonamides and later the antibiotics have all but eliminated the need for surgical intervention in acute suppurative otitis media and mastoiditis when patients are under the observation of competent otologists. There remains a group of patients, however, who have been treated inadequately by less competent physicians. A clinical picture of masked evidence of disease in which it is difficult for the most competent observer to decide upon rational treatment is encountered. There is evidence that in this group of patients chronic suppurative otitis and mastoiditis will continue to demand well-directed surgical intervention.

The serious complications of suppurations of the temporal bone are seldom encountered now. The younger otologic surgeons may never have the thrill of carrying out lifesaving measures that we older surgeons have had. Meningitis, infection of the blood stream and abscess of the brain secondary to suppuration of the temporal bone were always great problems and brought out the best efforts of serious-minded surgeons. Many lives were saved despite the odds against successful treatment.

The younger otologic surgeons, however, can have the thrill of restoring hearing in well-selected cases of otosclerosis because of the foundation work of Holmberg, Lempert and others. Improvement in technic may still be expected to extend the use of such surgical procedures. Then too, destructive operations of the labyrinth in carefully selected cases of Ménière's disease in which there has been little or no response to medical treatment should restore the patient to a useful life. Facial palsy resulting from mastoid operations has

been corrected in many patients as a result of the research work of Ballance and Duell.

A third improvement has taken place. A new science has developed within the specialty of otology which is designated as audiology. For the otologist to be a satisfactory audiologist he must have information and training that were not available to the older otologists. Knowledge of physics, chemistry and electronics has become so important in the estimation of hearing loss that to be an adequate diagnostician it is imperative that the otologist be trained in these sciences for the clinical application of the data obtained relevant to the problems encountered.

The tuning-fork tests still are important as is estimation of the emotional effect upon the patient of any hearing loss in a given individual. In many persons the data derived from clinical tests of hearing by scientific instruments of precision do not present the whole problem of diagnosis. It requires keen clinical acumen and judgment to interweave all the data into an accurate diagnosis. It is a challenge that calls for best efforts.

The older methods of treatment of deafness left much to be desired. To appease patients, otologists were often forced to use methods of treatment they knew in their hearts were not helpful. Occasionally an acute toxic neuritis causing deafness could be treated satisfactorily but the combined type of deafness and the senile type were not amenable to treatment. Within the past quarter of a century the development of aids for hearing by utilizing the science of electronics has made great strides. The rehabilitation resulting from the use of such aid has given relief and comfort to many patients. One seldom encounters patients now who remain adverse to the use of a hearing aid any more than the ophthalmologists encounter those who are adverse to the use of corrective lenses for their vision. More successful aids may rightfully be expected.

It expressly was understood that my remarks were to be brief, and to that end I shall not attempt to make a forecast of what the future holds in store. It can be said, however, that many problems will be encountered and solved and that otology can look forward to a glorious future, as it is evident to us older otologists that this specialty is in good hands because the otologists of today and tomorrow are to be well trained in the clinical and research aspects of the specialty.

MAYO CLINIC.

LXV

A NEW TEST FOR COCHLEAR RESERVE IN THE SELECTION OF PATIENTS FOR FENESTRATION SURGERY

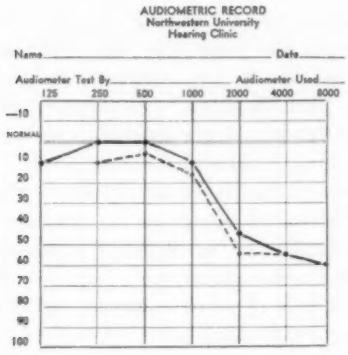
JAMES F. JERGER, M.A.

EVANSTON, ILL.

One of the most important problems facing the fenestration surgeon is the quantitative prediction of the postoperative hearing level. Theoretically, this postoperative threshold is equal to the sum of two factors; first, "conductive deficit" of 20 to 30 db, due to the absence of an ossicular chain,[1] and second, the amount of sensori-neural loss or "secondary nerve degeneration" present in the operative ear.

Under the assumption that the conductive deficit is constant for all patients, the problem of prediction is simply the problem of making a quantitative estimate of the sensori-neural loss. Previously advanced methods for making this estimate have been based on two measures, each of which is assumed to be a valid index of sensori-neural loss. One measure is bone conduction audiometry, and its use is best exemplified by the predictive method of Shambaugh.[9] The other measure, the speech discrimination or PB score, has been used most extensively by Walsh.[10]

The possibility of a third approach to the prediction problem was suggested to us by the fact that in recent years several investigators[3, 4, 5, 8, 11] have recommended the use of recruitment tests as a criterion for the selection of surgical cases. Until now, however, this approach has apparently been limited to dichotomous classification. In other words, the absence of recruitment has been interpreted as indicating suitability for surgery, and the presence of recruitment in any amount as contraindication to surgery. The purpose of the present study was to determine whether the *amount* of recruitment in the operative ear could be used to make a quantitative prediction of the postoperative threshold.



A

DL DIFFERENCE RECORD					
	250	500	1000	2000	4000
DL at 10 db	3.5	3.0	2.1	1.4	1.2
DL at 40 db	2.3	2.0	1.6	1.3	1.2
Diff.	1.2	1.0	.5	.1	0

B

Fig. 1.—Typical example of the relationship between the DL difference and the amount of sensori-neural loss in the recruiting ear. Chart A, air and bone audiogram of pure perceptive loss. Chart B, DL difference record. As the amount of sensori-neural loss increases, the DL difference decreases.

At Northwestern we have been concerned for some time with the development of a recruitment test capable of measuring not only the presence or absence of recruitment but its relative degree. The result of this research has been the "DL Difference Test,"[6] a test utilizing certain aspects of the methods previously proposed by Lüscher[7] and Denes and Naunton.[2] In this test the difference limen, or just noticeable difference, for a change in sound intensity is measured at two levels above threshold, 10 and 40 db. It has been found that the magnitude of the *difference* between these two difference limens, or DLs, reflects the degree of recruitment at the frequency being tested. Specifically, as the degree of recruitment increases, the magnitude of the difference decreases. Figure 1 illustrates this phenomenon in the case of a perceptive loss of unknown etiology. The audiograms shows normal hearing at 250 and 500 cps, then a gradually increasing loss reaching 55 db at 4000 cps. Reger's equal loudness contour test demonstrated recruitment at 2000 and 4000 cps. The bottom row of chart B, the DL difference record, shows how the *difference* between the two DLs decreases from a difference of 1.2 at 250 cps to a difference of zero at 4000 cps.

This finding suggested that the test might prove useful in estimating the amount of sensori-neural loss in the otosclerotic ear from which a prediction of the postoperative level could be made. In other words, if increasing sensori-neural loss correlates with decreasing DL difference, it should be possible to work backwards and predict the sensori-neural loss from the DL difference.

Accordingly, the test was first standardized on non-otosclerotic perceptive losses with recruitment, in order to establish an exact relationship between size of DL difference and sensori-neural loss in decibels, then applied to 55 preoperative fenestration cases to determine whether or not prediction of the postoperative threshold could be successfully made.

PROCEDURE

Standardization of the DL difference test was achieved by administering it to 18 cases otologically diagnosed as pure perceptive losses of varying etiology. The criterion for the presence of recruitment at a particular frequency was the presence of an abnormally small DL difference, using norms previously determined for normal

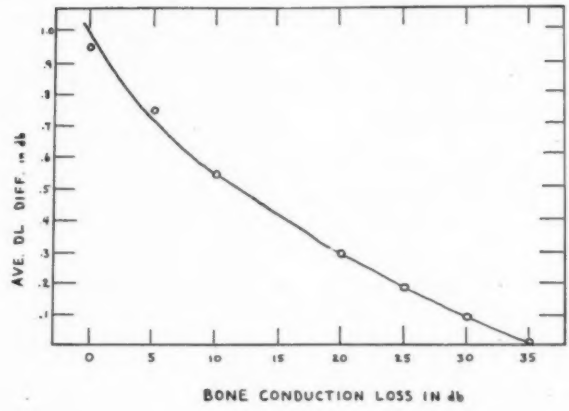


Fig. 2.—Relationship between DL difference and bone conduction loss in 18 pure perceptive losses with recruitment (pooled averages for 500, 1000, and 2000 cps).

hearers.[6] In the majority of cases it was possible to confirm this criterion with either Fowler's alternate binaural loudness test or Reger's monaural equal loudness contour method.

Thus at each frequency, on each patient, two measurements were made; first, the decibel loss by bone conduction, and, second, the magnitude of the DL difference in decibels. The DL difference was then averaged for each 5 db level of bone loss. Since statistical analysis showed that these averages were equivalent for a particular bone loss at each of the three frequencies, 500, 1000, and 2000 cps, the data for each frequency were pooled to form an over all average difference for each level of bone loss. Figure 2 shows the relationship between this average DL difference and the bone conduction loss. The curve of best fit through these averages was used to construct Table I, which became the basis for the prediction of the fenestration result. The table was derived from Figure 2 by determining the average DL difference corresponding to each 5 db level of bone loss using the curve rather than the observed average.

TABLE I.—RELATIONSHIP BETWEEN DL DIFFERENCE AND PREDICTED SENSORI-NEURAL LOSS (SECONDARY NERVE DEGENERATION) IN DB.

DL DIFFERENCE	PREDICTED SENSORI-NEURAL LOSS
1.00	0
.75	5
.50	10
.40	15
.30	20
.20	25
.10	30
0	35 or greater

The rationale for the use of Table I in predicting the postoperative level is simple. If, on the average, a 15 db loss by bone conduction is accompanied by a DL difference of .40 db in pure perceptive losses with recruitment, then it is reasonable to predict that the "true" bone threshold in an otosclerotic ear lies at 15 db if a DL difference of .40 db is obtained, assuming that the sensori-neural loss in otosclerosis is accompanied by recruitment.

The DL difference test was next administered to 55 preoperative fenestration patients. All cases were drawn from the routine patient load of Dr. G. E. Shambaugh Jr. and his associates over a 6 month period. The test was administered at the three frequencies, 500, 1000, and 2000 cps. Prediction of the postoperative level at each of these frequencies was made by adding 25 db (assumed conductive deficit)[9] to the sensori-neural loss predicted from Table I. For example, if a patient had a DL difference of .30 db at a particular frequency, his predicted postoperative level at that frequency would be 20 plus 25 or 45 db. If the DL difference was 1.00 db or greater, the predicted result would be simply 25 db, the optimum result by this method of prediction.

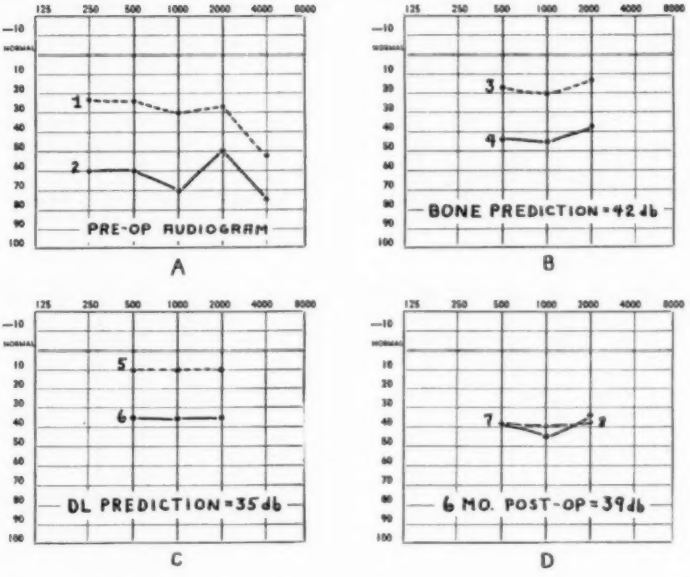


Fig. 3.—Bone prediction, DL prediction, and 6 month postoperative level on a typical fenestration case. Line 1, preoperative bone; line 2, preoperative air; line 3, preoperative bone corrected for Carhart Notch; line 4, prediction of postoperative lavel based on corrected bone; line 5, neural-sensory loss predicted by DL difference test; line 6, postoperative level predicted by DL difference; line 7, postoperative level at 6 months; line 8, prediction of postoperative level based on average of bone and DL predictions.

In the evaluation of the predictive accuracy of this method, the criterion for the actual postoperative result was chosen as the average of the two air audiograms obtained at three and six months postoperatively.

RESULTS

The analysis of the predictive ability of the DL difference test is most meaningful in terms of a comparison between the DL prediction and the prediction based on bone conduction audiometry as described by Shambaugh.[9] Briefly, Shambaugh's prediction is achieved

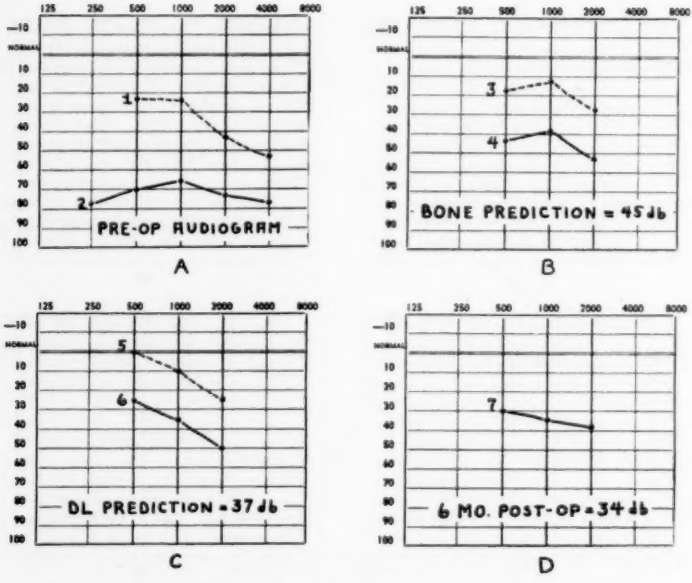


Fig. 4.—Bone prediction, DL difference, and 6 months postoperative level on a case in which the DL prediction was substantially more accurate than the bone prediction. Designation of lines same as in Figure 3.

by adding 25 db to the bone threshold after it has been corrected for the "Carhart Notch." In the present 55 cases three separate preoperative bone audiograms were made on different days, and the prediction was based on the average of these three audiograms. Contralateral masking was used in all bone tests.

Figure 3 shows both predictions and the ultimate postoperative level on a typical case. Chart A shows the preoperative audiogram on the ear to be fenestrated. In chart B the preoperative bone at 500, 1000, and 2000 cps has been corrected for the Carhart Notch (raised 5 db at 500, 10 db at 1000, and 15 db at 2000 cps). Line 4 in this chart is the predicted result by Shambaugh's method (i.e., a line 25 db below the corrected bone audiogram). Thus the predition of the pure tone average for the three speech frequencies is 42 db.

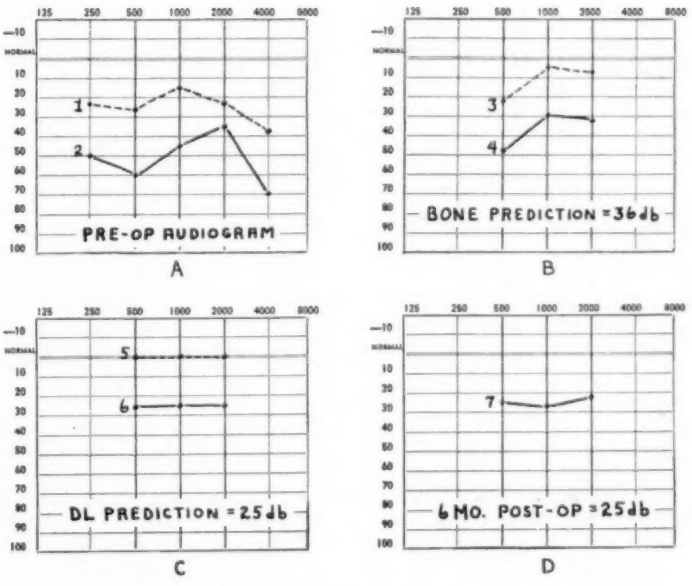


Fig. 5.—Bone prediction, DL prediction, and 6 month postoperative level on a case in which the DL prediction was more accurate than the bone prediction. Designation of lines same as in Figure 3.

Chart C shows the prediction based on the DL difference test. Line 5 is the neural-sensory loss predicted from Table I. This case showed a DL difference of .50 db at each frequency. Hence the predicted neural-sensory loss at each frequency is 10 db. Line 6 is the predicted result according to the DL difference test method (i.e., a line 25 db below the predicted neural-sensory loss). Therefore, by this method, the prediction of the pure tone average for the speech frequencies is 35 db. Line 7 in chart D shows the postoperative level attained. Line 8 is the prediction that would have resulted from averaging the bone and DL predictions. Figures 4 and 5 show cases in which the DL prediction was considerably more accurate than the bone prediction. On the other hand, Figure 6 shows a case in which the bone prediction was more accurate.

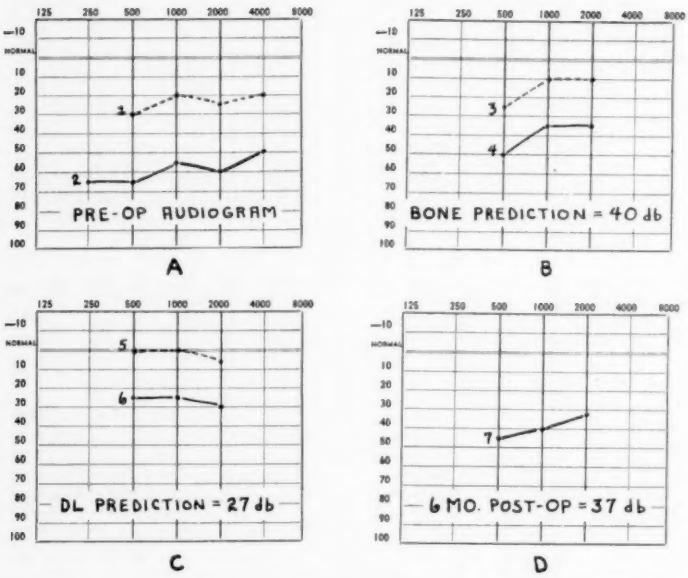


Fig. 6.—Bone prediction, DL prediction, and 6 months postoperative level on a case in which the bone prediction was superior to the DL prediction. Designation of lines same as in Figure 3.

Of the 55 cases tested there were 30 in which both methods predicted a pure tone average of 29 db or better. Since all of these cases reached a postoperative level of 29 db or better they are of little value for comparative purposes. In the remaining 25 cases, one or both methods predicted a result poorer than 29 db. One of these cases showed no postoperative gain and was regarded as a surgical failure. Analysis of the remaining 24 cases demonstrated that the DL prediction was, on the average, slightly superior to the bone conduction prediction. Table II shows the number of times that both methods were in error by more than 5 db and by more than 10 db for the 500-2000 average. Neither method was ever in error by more than 15 db.

TABLE II.—NUMBER OF TIMES EACH METHOD WAS IN ERROR BY MORE THAN 5 DB AND BY MORE THAN 10 DB (24 PATIENTS).

	NO. OF TIMES IN ERROR BY MORE THAN 5 DB	NO. OF TIMES IN ERROR BY MORE THAN 10 DB
DL prediction	5	1
Bone prediction	11	4

The superiority of the DL prediction is not statistically significant for errors greater than 5 db, but it is highly significant for errors greater than 10 db. However, the use of statistical comparisons in this kind of categorization is questionable.

Another method of evaluating these predictive methods is by a comparison of the number of times each method was more accurate than the other by a substantial amount. In these 24 cases there were just two in which the bone prediction was more accurate than the DL prediction by more than 5 db. There were six cases in which the DL prediction was more accurate than the bone prediction by more than 5 db. In one of these cases the DL difference was closer by 11 db.

COMMENT

These results have important implications for the fenestration surgeon since they show that in the sample studied it was possible to make a reasonably accurate quantitative estimate of the post-operative level purely on the basis of the recruitment phenomenon. The practical consequence of this finding is that eventually we should be able to place at the disposal of the fenestration surgeon a method of prediction which is independent of both bone conduction and speech audiometry. On the reasonable assumption that three tests are better than two, this should enable the fenestration surgeon to increase the precision of his preoperative predictions.

It must be emphasized, however, that in its present state of development the DL difference test is not sufficiently refined and simplified for routine clinical use in this fashion. Further research is essential before the test can be used successfully by the surgeon or his office audiometrist.

SUMMARY

The DL difference test, a measure of the degree of recruitment present in the operative ear, was administered to 55 preoperative fenestration cases. The prediction of the postoperative level (6 months) based on the test was slightly superior to the prediction based on bone conduction audiometry (Shambaugh method).

Although further research is necessary before the test will be sufficiently simplified for routine clinical use, the results of this study have practical significance for the fenestration surgeon. By utilizing the recruitment phenomenon he should be able to supplement his present clinical tools with an additional predictive method independent of bone and speech audiometry.

ACKNOWLEDGEMENT

It is a pleasure to acknowledge the helpful guidance of Dr. R. Carhart and Dr. G. E. Shambaugh, Jr., throughout the progress of this research.

NORTHWESTERN UNIVERSITY.

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LXVI

A LABORATORY ASSESSMENT OF HEARING ACUITY FOR VOICE SIGNALS AGAINST A BACKGROUND OF NOISE

KINSEY M. SIMONTON, M.D.

AND

LEROY D. HEDGECOCK, PH.D.

ROCHESTER, MINN.

We have been responsible for several years for evaluation of the hearing of a group of airline pilots, and for making recommendations concerning their continued employment by the airline. The established criteria, based on the pure-tone-threshold audiogram by air conduction, seemed inadequate for the purpose. The studies to be reported were undertaken for our own information in solving this problem.

The problem was originally set up as a comparison of thresholds for speech signals in quiet and in noise.[1] Comparisons were made between the pure-tone threshold in quiet (35 decibels) and the thresholds for speech signals using the Harvard spondaic words and interconnected discourse delivered from phonograph recordings both in free field and under earphones in quiet (35 decibels) and noise (98.5 decibels). Four groups of individuals were tested. These were (1) normal-hearing subjects, (2) patients with middle-ear conduction deafness, (3) patients with perception deafness due to a variety of causes and (4) patients with cochlear end-organ deafness due to Ménière's disease.

The following conclusions were derived from data collected and were reported by one of us (Hedgecock) at a meeting of The American Speech and Hearing Association.

1. The average threshold for pure tones for the three speech frequencies between 500 and 2,000 cycles per second correlates closely

with the speech-reception threshold when both tests are taken in quiet condition.

2. The speech-reception threshold for normal ears varies with the intensity of the masking noise and is approximately 20 decibels less than the masking noise.

3. Speech-reception thresholds are approximately the same when the signal is applied to one or both ears. Therefore, for signals applied to both ears, the threshold is primarily a function of the better-hearing ear.

4. The effect of masking noise is greater when applied to the ear receiving the signal than when applied to the opposite ear.

5. Defects of middle-ear conduction of sound protect the ear against masking noise to a degree which approximates the impediment to sound conduction.

6. Defects of perception of sound protect the ear against masking noise but to a lesser degree than defects of middle-ear conduction of sound.

7. The threshold of hearing for speech signals in noise is neither improved nor impaired by experience in listening in noise.

8. The data reported do not provide a basis for determining the efficiency of hearing of speech signals of above-threshold intensity in the presence of noise. Studies of ability of defective ears to discriminate speech at intensities above threshold in the presence of masking noise are needed.

Lightfoot and co-workers[3] report similar conclusions from their data derived from a thoroughly studied series of individuals.

Our study was extended to include determinations of ability to discriminate speech signals in quiet and in noise.

METHODS USED

The masking noise used for these studies was set up to approximate the over-all intensity and the frequency characteristic of noise present in the cockpit of an airplane in flight. Determinations of the over-all frequency were made with the General Radio noise level meter and General Radio frequency analyzer in the cockpits of three airplanes operating at normal cruising speeds. Two of the airplanes

had two motors each; the third had four motors. Three distinct models were represented. Each airplane was in the regular service of a commercial airline. The average over-all values for noise and individual frequencies in these three airplanes formed the basis for the masking noise.

The masking noise consisted of a mixture of white noise, 60 cycle harmonics and 112 cycle tones. This was delivered by loud-speaker in a sound-insulated room. The subject under test was seated approximately 6 feet (183 cm) from the loud-speaker. The over-all intensity of the masking noise at the subject's seat was 98.5 decibels.* Maximal intensity for a single frequency was 97 decibels at 112 cycles per second. Intensities gradually decreased to 71 decibels at 620 cycles per second, with peaks at the harmonics of 60 and 112 cycles per second. Frequency components were present up to 5,000 cycles per second although at lesser intensities.

The validity of this masking noise is attested by the report of Karlin and co-workers[2] that the ability to listen in noise is independent of differences in spectra of the interfering noise.

Tests were conducted in the following manner: A pure-tone audiogram was made in a quiet, sound-insulated room. The level of ambient noise in this quiet room was found to be approximately 35 decibels. The speech-reception threshold for the Harvard spondaic word lists was first determined without additional noise. The test words were delivered by loud-speaker from a phonograph recording. The same test then was repeated in the presence of the masking noise. The Harvard phonetically balanced word lists were then delivered by loud-speaker from phonograph recordings in the quiet room and in the presence of the masking noise at signal intensities which the listener indicated as the most comfortable level.

CONTROLS

Twelve young adults who heard normally were tested as controls (table). Inspection of the average values for the control group

*The physicist's value for 0 decibel is customarily 0.0002 dyne per square centimeter. The standard audiometer is so calibrated that 0 decibel has a value of approximately 9 decibels re 0.0002 dyne per square centimeter in speech frequencies of 500 to 2,000 cycles per second. In this discussion pure-tone audiograms will be reported according to the calibration scale of the audiometer. All other references to the decibel will be in relation to a value of 0.0002 dyne per square centimeter for 0 decibel.

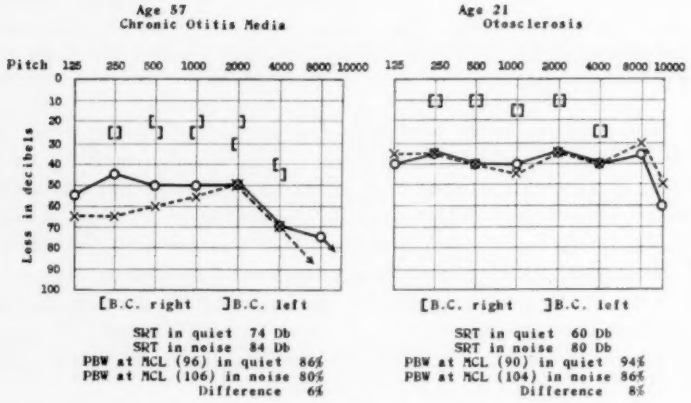
Age 57
Chronic Otitis Media
Age 21
Otosclerosis
Pitch 125 250 500 1000 2000 4000 8000 10000
Loss in decibels
[B.C. right]B.C. left
SRT in quiet 74 Db
SRT in noise 84 Db
PBW at MCL (96) in quiet 86%
PBW at MCL (106) in noise 80%
Difference 6%
SRT in quiet 60 Db
SRT in noise 80 Db
PBW at MCL (90) in quiet 94%
PBW at MCL (104) in noise 86%
Difference 8%

Fig. 1.—Hearing acuity against a background of noise: conduction deafness.

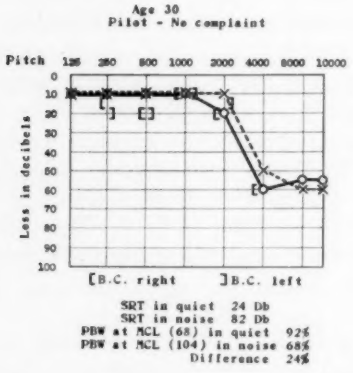
Age 30
Pilot - No complaint
Pitch 125 250 500 1000 2000 4000 8000 10000
Loss in decibels
[B.C. right]B.C. left
SRT in quiet 24 Db
SRT in noise 82 Db
PBW at MCL (68) in quiet 92%
PBW at MCL (104) in noise 68%
Difference 24%

Fig. 2.—Hearing acuity against a background of noise: perception deafness.

TABLE 1.
HEARING ACUITY FOR VOICE SIGNALS AGAINST A BACKGROUND OF NOISE. 12 NORMAL SUBJECTS.

S.R.T. in quiet (35 Db)	20 db
S.R.T. in noise (98.5 Db)	80 db
P.B.W. at M.C.L. (66-70 Db) in quiet	98-100%
P.B.W. at M.C.L. (102-108 Db) in noise	78-88% (Av. 83)
Difference, average	16%

indicated that the threshold of hearing for speech signals in quiet surroundings (20 decibels) approximated the average thresholds for pure tones in the speech frequencies. The threshold for speech reception delivered against a masking noise of 98.5 decibels was 60 decibels higher than for the same signals delivered in the quiet room in which the level of ambient noise was approximately 35 decibels. Thus, for normal ears the increase in threshold approximated the increase in masking noise present in the two conditions of the test.

The intensities selected as most comfortable by members of the control group varied only 4 decibels for tests conducted in quiet and 6 decibels for tests conducted in noise. The percentage of words repeated correctly varied from 98 to 100 per cent, averaging 99 per cent, for tests in quiet. For tests in noise the percentage of words repeated correctly varied from 78 to 88 per cent with an average value of 83 per cent. Thus the loss of discrimination by normal subjects resulting from listening in the presence of the masking noise averaged 16 per cent.

It is of interest to note that for tests in noise the normal subjects who selected the higher intensities for the speech signal made better scores than the subjects who selected lower intensities. This compares favorably with the results reported by Abrams and associates[1] of discrimination scores for normal listeners in simulated airplane noise of 115 decibels intensity. The scores varied from 19 per cent at a speech-to-noise ration of −10 decibels to 94 per cent at a speech-to-noise ratio of +15 decibels.

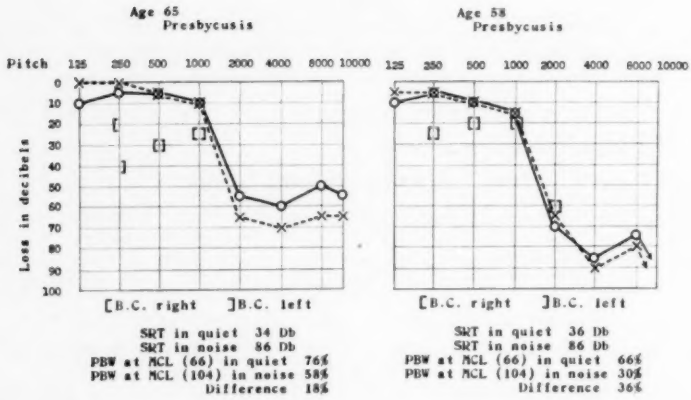
Age 65
Presbycusis
Pitch 125 250 500 1000 2000 4000 8000 10000
Loss in decibels
0 10 20 30 40 50 60 70 80 90 100
[B.C. right]B.C. left
SRT in quiet 34 Db
SRT in noise 86 Db
PBW at MCL (66) in quiet 76%
PBW at MCL (104) in noise 58%
Difference 18%
Age 58
Presbycusis
125 250 500 1000 2000 4000 8000 10000
[B.C. right]B.C. left
SRT in quiet 36 Db
SRT in noise 86 Db
PBW at MCL (66) in quiet 66%
PBW at MCL (104) in noise 30%
Difference 36%

Fig. 3.—Hearing acuity against a background of noise: perception deafness.

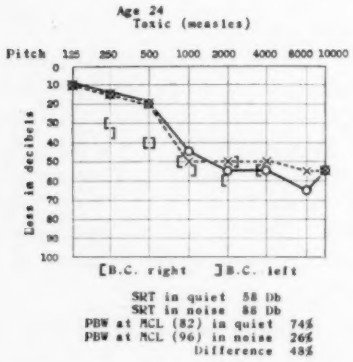
Age 24
Toxic (measles)
Pitch 125 250 500 1000 2000 4000 8000 10000
Loss in decibels
0 10 20 30 40 50 60 70 80 90 100
[B.C. right]B.C. left
SRT in quiet 58 Db
SRT in noise 86 Db
PBW at MCL (82) in quiet 74%
PBW at MCL (96) in noise 26%
Difference 48%

Fig. 4.—Hearing acuity against a background of noise: perception deafness.

DEFECTIVE EARS

Data are presented as records of individual patients rather than as a statistical analysis. For a statistical analysis of similar cases see the report of Tonndorf.[5]

Conduction Deafness.—Records of 2 patients presenting middle-ear conduction deafness are shown in Figure 1. When compared to the normals these show marked increase in speech-reception threshold in quiet, but little or no increase in the speech-reception threshold in noise. The most comfortable level in quiet is higher than that selected by normals, while the comfort level in noise compares with that selected by normals. Discrimination is less efficient than that for normals in quiet, but equally efficient in noise. These findings confirm existing opinion that middle-ear conduction deafness presents no handicap to discrimination of speech in an environment of noise.

Perception Deafness.—The records of 6 patients will be shown. The first (Fig. 2) is that of an airplane pilot who had no complaint referable to hearing. The pure-tone audiogram shows abrupt loss for frequencies above 2,000 cycles per second. Speech-reception thresholds and comfort levels closely approximate those of normal subjects, yet discrimination is reduced in quiet and in noise, the decrease due to noise being greater than for normals. This man had long experience listening in noise.

The records of 2 patients with abrupt hearing loss for frequencies above 1,000 cycles per second due to presbycusis showed remarkable similarity in pure-tone thresholds, speech-reception thresholds and most comfortable levels (Fig. 3*a* and *b*). The speech-reception threshold in quiet is 14 decibels greater than that for the controls, while speech-reception threshold in noise is 6 decibels greater than for the controls. Comfort levels are similar to those for the controls. The 2 subjects show marked difference in ability to discriminate speech both in quiet and in noise. This difference is not explained by available data.

The record of a young woman, aged 24, who suffered hearing loss during an attack of measles at the age of 4 years is shown in Figure 4. Her pure-tone audiogram shows a sharp drop for frequencies above 500 cycles per second. The speech-reception threshold is increased 38 decibels in quiet, 8 decibels in noise. The most

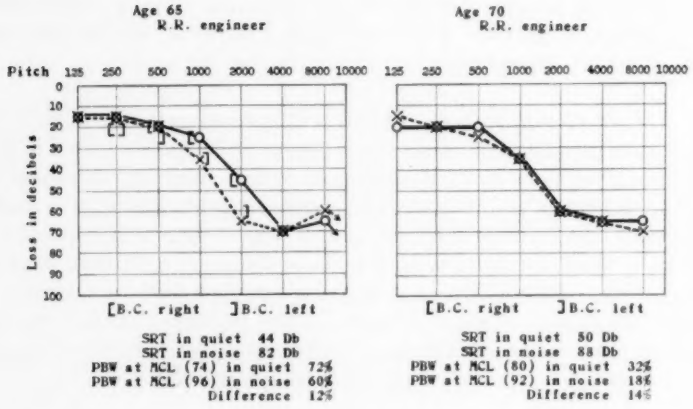
Age 65
R.R. engineer
Age 70
R.R. engineer
Pitch 125 250 500 1000 2000 4000 8000 10000
Loss in decibels
0 10 20 30 40 50 60 70 80 90 100
[B.C. right]B.C. left
SRT in quiet 44 Db
SRT in noise 82 Db
PBW at MCL (74) in quiet 72%
PBW at MCL (96) in noise 60%
Difference 12%
SRT in quiet 50 Db
SRT in noise 88 Db
PBW at MCL (80) in quiet 32%
PBW at MCL (92) in noise 18%
Difference 14%

Fig. 5.—Hearing acuity against a background of noise: perception deafness.

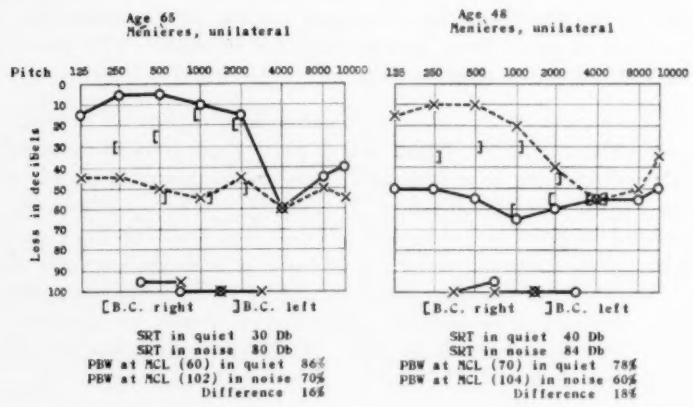
Age 65
Menieres, unilateral
Age 46
Menieres, unilateral
Pitch 125 250 500 1000 2000 4000 8000 10000
Loss in decibels
0 10 20 30 40 50 60 70 80 90 100
[B.C. right]B.C. left
SRT in quiet 30 Db
SRT in noise 80 Db
PBW at MCL (60) in quiet 86%
PBW at MCL (102) in noise 70%
Difference 16%
SRT in quiet 40 Db
SRT in noise 84 Db
PBW at MCL (70) in quiet 78%
PBW at MCL (104) in noise 60%
Difference 18%

Fig. 6.—Hearing acuity against a background of noise: unilateral Ménière's disease.

comfortable level in quiet is high, but in noise at 96 decibels is lower than that selected by the controls. Discrimination is poor in quiet despite the strong signal selected. Discrimination in noise is very poor. Part of this loss is due to the low signal intensity which is 2.5 decibels less than the masking noise.

Two patients, railroad engineers of similar ages, both stated that hearing function was perfect in the noise of an engine. Their threshold audiograms for pure tones were similar. Marked dissimilarity was shown by speech tests. One (Fig. 5*a*) showed that the speech-reception threshold was moderately raised in quiet and slightly raised in noise, and that the most comfortable level was increased moderately in quiet but decreased in noise. Discrimination in quiet was very good (72 per cent) and loss of discrimination incident to noise was less than that for controls despite the unfavorable noise-to-signal ratio. The other test (Fig. 5*b*) showed much greater rise in speech-reception threshold in quiet and in noise, large rise in comfort level in quiet and marked lowering in comfort level in noise. Discrimination was very poor in quiet (32 per cent) in spite of adequate signal intensity. The discrimination loss due to noise (14 per cent) is less than that for the controls, but discrimination in noise is below the level of practical hearing. These two patients again present unexplained differences in ability to discriminate spoken words.

Cochlear End-organ Deafness.—The records of 2 patients presenting findings of unilateral Ménière's disease are shown in Figure 6*a* and *b*. In each patient the threshold of hearing for the better ear was deficient for the high tones. The principal characteristic of both threshold and discrimination tests is the similarity to cases of perceptive deafness. This suggests that in free-field testing of patients with unilateral Ménière's disease the results of threshold and discrimination tests are a function of the better ear, and that distortion due to recruitment of loudness in the diseased ear does not disturb the function of the better ear.

The records of 2 patients presenting findings of bilateral Ménière's disease are shown in Figure 7*a* and *b*. These patients reveal marked rise in speech-reception threshold both in quiet and in noise. The most comfortable level also is high in quiet despite evidence of a high degree of loudness recruitment in each ear. The most comfortable level in noise is at the lower level selected by the controls.

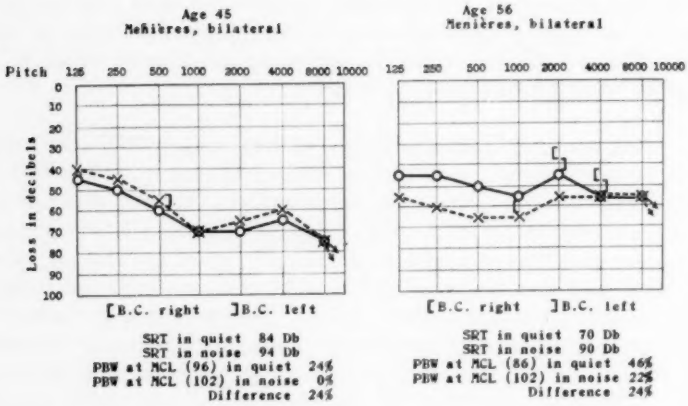
Age 45
Meñières, bilateral
Pitch 125 250 500 1000 2000 4000 8000 10000
Loss in decibels
0 10 20 30 40 50 60 70 80 90 100
[B.C. right]B.C. left
SRT in quiet 84 Db
SRT in noise 94 Db
PBW at MCL (96) in quiet 24%
PBW at MCL (102) in noise 0%
Difference 24%
Age 56
Menières, bilateral
125 250 500 1000 2000 4000 8000 10000
[B.C. right]B.C. left
SRT in quiet 70 Db
SRT in noise 90 Db
PBW at MCL (86) in quiet 46%
PBW at MCL (102) in noise 22%
Difference 24%

Fig. 7.—Hearing acuity against a background of noise: bilateral Ménière's disease.

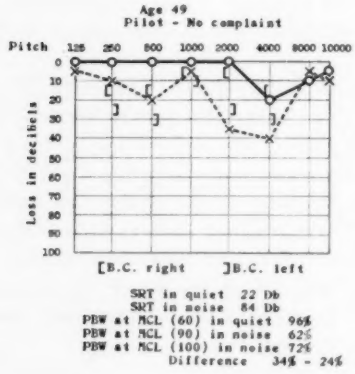
Age 49
Pilot - No complaint
Pitch 125 250 500 1000 2000 4000 8000 10000
Loss in decibels
0 10 20 30 40 50 60 70 80 90 100
[B.C. right]B.C. left
SRT in quiet 22 Db
SRT in noise 84 Db
PBW at MCL (60) in quiet 96%
PBW at MCL (90) in noise 62%
PBW at MCL (100) in noise 72%
Difference 34% - 24%

Fig. 8.—Hearing acuity against a background of noise: effect of variations in signal intensity.

Discrimination is poor in quiet, and the loss incident to masking noise results in serious impairment of useful hearing.

The final record is shown to demonstrate the effect of unfavorable signal-to-noise ratio on discrimination of speech (Fig. 8). The subject selected a most comfortable level of 90 decibels, which is 8.5 decibels less intense than the masking noise. At this intensity the discrimination score is 62 per cent. The discrimination score is improved 10 per cent by a 10 decibel increase in signal intensity.

COMMENT

The current interest in loss of hearing due to noise in industry poses several questions. Among these questions are the following.

1. What is the influence of noise on perception of speech by defective ears? The answer depends upon the type of hearing loss present. The efficiency of the ear deafened because of impaired middle-ear conduction is not adversely affected by noise. The function of the perception-deafened ear in noise is reduced in varying degrees. General rules cannot be derived from the data studied. The efficiency in noise of the patient with unilateral cochlear deafness is a function of the better-hearing ear. Patients with bilateral cochlear deafness possess very poor hearing function in noise.

2. What degree of hearing impairment should disqualify a worker for employment in an environment of noise? This depends on the necessity for effective hearing for the safe and efficient conduct of the work, as well as on the factors discussed with question 1.

3. What is the relation between threshold of hearing in quiet and discrimination of speech in noise? The threshold is of little significance unless considered in conjunction with the type of deafness and the shape of the curve for pure-tone threshold. In uncomplicated middle-ear conduction deafness, ability to discriminate in noise is not related to thresholds in quiet. Cases of perception deafness studied in this series indicate increasing loss of ability to discriminate in noise with shift of threshold losses toward the lower frequencies of the speech range. Wide individual variations in ability to discriminate in noise which were not related to threshold variations were noted. Cases of bilateral cochlear deafness studied all revealed severe losses both for threshold and for discrimination in noise.

We are familiar with the difficulty experienced by persons with perception deafness in hearing conversation in noisy places. On the other hand we have observed individuals who, despite perception losses by tests of threshold in quiet suroundings, maintained that their hearing efficiency was normal in the cockpit of an airplane or the cab of a locomotive. How can we resolve such opposite observations? Does experience in listening in noise increase the efficiency of hearing in noisy environments? Our observations on the subjects studied suggest that experience did not increase efficiency of hearing in noise and that self-evaluation of ability to hear in noise did not correlate with ability to discriminate the test signals used.

CONCLUSIONS

From comparison of ability to discriminate speech signals in environments of quiet and of noise, the following conclusions are suggested:

1. Both the type of hearing loss and the threshold level are required to estimate the ability to discriminate in noise.

2. Loss of hearing due to impaired middle-ear conduction does not reduce discrimination of speech in environments of noise.

3. Loss of hearing due to impaired perception of sound resulted in moderate loss of discrimination in noise. Wide individual variations in discrimination were demonstrated.

4. In unilateral cochlear deafness, distortion due to recruitment in the poorer ear did not affect the discrimination scores from the better ear either in quiet or in noise.

5. Bilateral cochlear deafness resulted in severe loss of discrimination both in quiet and in noise.

6. A favorable ratio of intensity between signal and noise is necessary for efficient hearing in noise.

7. The self-estimates of ability of certain perception-deafened persons to hear in noise were not confirmed.

MAYO CLINIC.

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LXVII

EXPERIMENTAL OBSERVATIONS ON SOUND CONDUCTION IN THE MIDDLE AND INNER EAR

H. G. Kobrak, M.D.

Chicago, Ill.

The experimental methods and the motion picture recordings which are demonstrated here today are the extension of former work on sound conduction in the ear. In the previous experiments optical recordings were made by means of small mirrors attached to the ear structures. This method was adequate for recordings of the eardrum and the ossicles.

When the demands for greater magnification increased and the recording of more delicate structures became desirable, the limit of the mirror technic was obviously reached. Especially the recording of the intracochlear fluids or of the gelatinous structures in the inner ear can be made only by methods which do not require the affixing of mirrors and do not necessitate instrumental contact of any sort.

The motion picture camera possesses many characteristic qualities which made it a favorable instrument of scientific investigation.

The method could be described as "microscopic-stroboscopic cinematography." In physiological acoustics the phenomena under observations are rapid and minute. Optical magnification available at present enlarges the structures sufficiently. The sensitivity of our present optical set-up is such that movements of 10 micra or more can be observed. This is the size of a leucocyte. By utilizing more powerful microscopes the optical system could be made more sensitive, if it becomes desirable. Most favorable for photographic recordings are relatively low impedance systems. In a low impedance system the amplitudes are large in response to a given stimulus. In contradistinction, a high impedance system responds with small amplitudes, a part of the stimulus resulting in stress variations and frictional forces. It is important to realize that the camera records

From the Division of Otolaryngology of The University of Chicago.

only the amplitude of vibration, it fails to show pressure changes or heat variations. The impedance of the eardrum has been measured by a number of workers. For the frequency range of our experiments the eardrum is undoubtedly a low impedance system. For the frequency around 800 the eardrum has an impedance as low as that of air. This fact is one basic reason why we consider the camera a good method for the study of the ear.

A second consideration in the discussion of the method is the rapidity of the phenomena. The rapidity can be overcome by "time microscopy." Either high speed camera recordings or photography under stroboscopic illumination can accomplish the deceleration of rapid vibratory phenomenon to a satisfactory content.

EXPERIMENTAL PART

The auditory end organ is enclosed in bone. In order to function properly the cochlea must have at least two areas which possess a high compliance. In the normal ear this is the oval and round window. The oval window is the end point of the ossicular chain. The round window is in direct contact with the air of the tympanic cavity. The conduction of sound through the eardrum, hammer, incus and stapes is known as ossicular sound conduction.

A descriptive term for the conveyance of sound energy through the eardrum and the air of the tympanic cavity was coined by Gustaf Alexander of Vienna who called it "aero-cochlear" sound conduction. The tympanic cavity is a sound conductor, its air contents having definite physical characteristics. The otologist has not been able to assign the middle ear spaces a clear role in sound conduction, first because air is invisible and does not appear as a structural entity in histological ear sections. Secondly, perhaps because the understanding of the physical qualities of the air bubble encased in the tympanic spaces is incomplete.

The attempt was made in our experiments to visualize the role of the intratympanal air in sound conduction. Two tracer substances were used to identify the vibrations of the air particles. One was cigaret smoke, the other minute fibers of cotton. Smoke consists of a multitude of microscopic white particles which under proper microscopic magnification and stroboscopic illumination can be observed and photographed during acoustic stimulation. Our preliminary results with this method can be summarized that the usage of

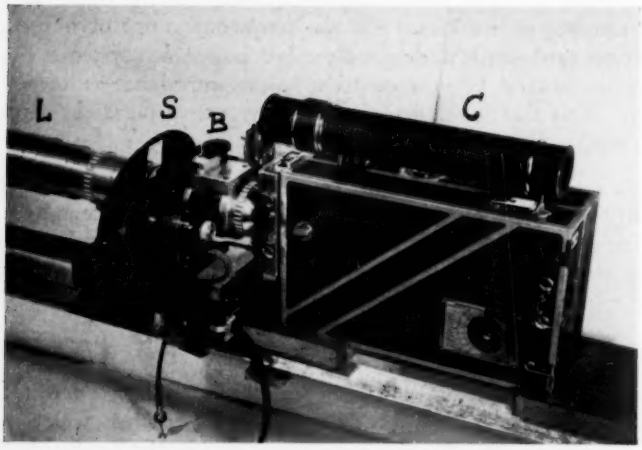

Fig. 1.—Macroscopic Stroboscopic Cinematography. The arrangement of Lens (L), Stroboscope (S), Beamsplit (B) and Camera (C) on the optical bench. Extension tubes of various length placed between lens and camera increase the magnification. Maximum manification used with extension tubes was an object to image ratio of 1:4. The Beamsplit permits simultaneous cinematographic recording of the sound stimulus (e.g. Cathode Ray Oscilloscope) and the response in the ear.

smoke particles as tracer substance is feasible. Investigations on intact eardrums and perforated eardrums have given confusing results and will not be discussed at this time. The use of minute cotton fibers as tracer substances is technically much easier and will be demononstrated by film recordings. It is hoped that by further development of the method the term "aero-cochlear sound conduction" ceases to be a theoretical concept and becomes a recognized phenomenon of physiological acoustics just as ossicular sound conduction is experimentally proven.

Using fresh human temporal bones, the following observations were made during acoustic stimulation.

1) The eardrum and the three ossicles execute acoustic vibrations which can be observed both macroscopically and microscopically under stroboscopic light.

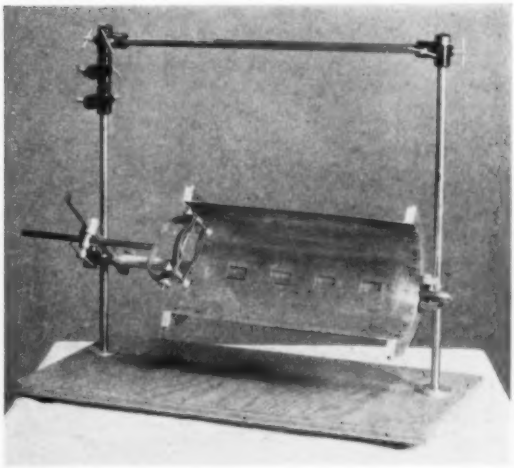

Fig. 2.—The Animal Carrier. The rigidity of the optical bench necessitates an arrangement by which the experimental animal can be moved freely in all directions. Gross focusing and framing is done by the adjustments of the carrier. Fine focusing and minute changes in framing of the picture are done on the microscope carrier. (See Fig. 3)

2) The stapes shows two types of vibrations as described first by Dahlmann and Bekesy. In addition to the regular vibrations around an axis parallel to the footplate was observed. In contradistinction to Bekesy's description of the phenomenon, a sudden change ("Umkippen") was not noticed, but a gradual change of axis was observed.

3) The phase difference between the oval and round window is evident in nearly all experiments. The phase difference between the stapes vibration and the oscillation of the round window membrane is approximately 180°.

4) On intense stimulation gliding motions were observed in the incudostapedial articulation. A phase difference is created by the articulations: The stapes is lagging slightly behind the eardrum vibrations.

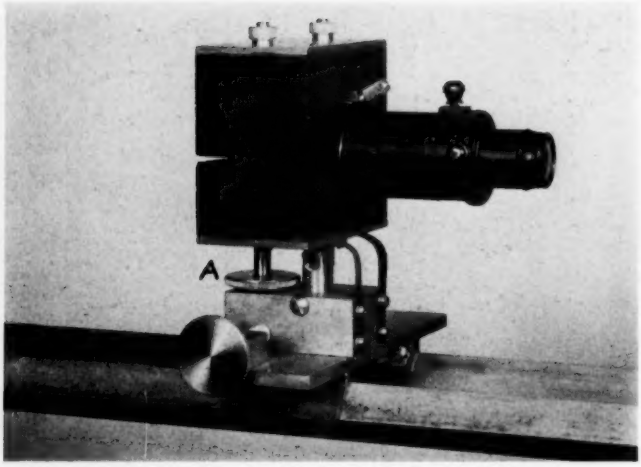

Fig. 3.—The microscope and carrier used for microscopic-stroboscopic cinematography. The tube length between objective and ocular can be changed. Thus, a variety of magnifications are obtainable. Fine up and down adjustments for framing are made on screw A, while lateral adjustment is made by screw B. Fine focusing is done by moving the microscope on the optical bench.

5) The amplitude of the round window membrane is larger than the amplitude of the stapes.

The problems of sound conduction can be readily studied in the rabbit. From an incision in the neck, an exposure of the bulla ossea is easily accomplished. An opening in the ventral wall of the bulla renders a good exposure of the eardrum, the whole ossicular chain, the promontory with both fenestrae and the two intratympanic muscles. No important middle ear structure has to be sacrificed in order to obtain this exposure. Sound is conducted into the outer ear canal. When an overthreshold sound stimulus is applied, the acoustic vibrations of the eardrum and of the ossicular chain become visible. The eardrum vibrates like a piston in its central part. The acoustic reflex of the intratympanic muscles is elicited regularly provided the frequency and the intensity of the signal are selected properly. Low pitched tones, e.g., 60 c.p.s. do not produce a reflex contraction.

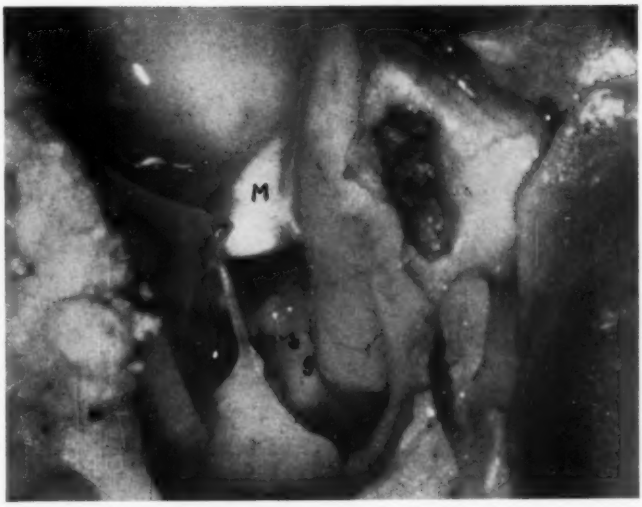

Fig. 4.—The Middle Ear of a Rabbit. During strong acoustic stimulation, both the Tensor Tympani Muscle (M) and the Stapedius (S) contract. Cinematographic recordings show a gliding motion of the stapes in the incudo-stapedial articulation in a posterior direction (arrow) during the contraction. The incus remains motionless. The eardrum does not show any motion during tensor contraction. On stroboscopic illumination one can see the muscle belly of the tensor tympani vibrate with the ossicular chain. The amplitudes of the eardrum at the umba can be measured by penoil tracings of different motion picture frames. The mode of vibration of the tympanic membrane can also be observed by this magnification.

Similarly, low intensities are subthreshold for the reflex. If one stimulates the ear with a loud tone of very low pitch (60-100 cps up to 120 db intensity) no reflex is elicited, while the ossicular chain executes definite acoustic vibrations. When a second tone of higher pitch is sounded a muscle contraction occurs. The following observations can then be made:

1) The tensor tympani contracts slowly. The time interval between onset of contraction and assumption of full tetanus has the order of magnitude of 0.25 second.

2) The amplitudes of acoustic vibrations are visibly diminished by the muscle contraction.

3) There is no visible movement of the eardrum due to the contraction of the tensor tympani.

4) Due to the contraction of the stapedius muscle there is a slow, gliding motion in the incudo stapedial articulation.

5) On strong stimulation, the tensor tympani muscle during its contraction, participates in acoustic vibrations. The tensor tympani in the rodent runs freely through the lumen of the middle ear cavity. In man, both muscles are encased in canals or subcavities. (Semicanalis m. tensor tympani and lumen of processus pyramidalis respectively.) The acoustic oscillations of the muscle belly may well be confined to species with unenclosed muscle belly.

In man, the following observations were made: 1) on persons with the intact eardrum, no movements can be seen which can be attributed to the contractions of the intratympanic muscles. An exception has been observed recently. A 45-year-old female patient with intact eardrum showed movements of the eardrum during acoustic stimulation; 2) patients with a perforation of the eardrum in the posterior superior quadrant may show an acoustic stapedius reflex. Movements of the stapedius tendon can be seen. No motion of the stapes is observed as long as the ossicular chain is intact. If, however, the incudo-stapedial articulation is interrupted or the incus absent, definite movements of the isolated stapes are produced by the acoustic stapedius contractions. The contraction of the stapedius muscle results in a tilting motion of the stapes in a posterior direction. In one case, the movement of the stapes was photographed and measured by screen tracings. It was found that the stapes was pulled backward by the acoustic contraction of the stapedius muscle a distance of 200 micra. It was calculated that the tilting motion of the stapes resulted in stretching the fibers of the anterior pole of the annular ligament from a 150 micra length in rest position to 210 μ length in the new position. The space between the two positions of the stapes footplate was calculated as 0.16 cubic millimeters. Secondary to this motion of the footplate, a displacement of the intralabyrinthine fluid must be assumed. There is no subjective sensation reported by the patients who show these static movements of the stapes during acoustic stimulation. It can be assumed that such a

static displacement of the perilymph is readily equalized with the opposite scala through the helicotrema.

The static displacement of intralabyrinthine fluid due to muscle contraction has been measured by other authors using different methods. Tsukamotu, using a glass capillary as labyrinth manometer in rabbits, found a displacement of 0.002 mm.[3] Wiggers interpreted some of his findings on cochlear microphonics as a fluid displacement due to stapes movements during stapedius contractions. In spite of the difference of the methods, these three observations are in fairly good agreement.

OBSERVATIONS IN THE INNER EAR

The conduction of sound in the inner ear can be studied by observation through windows, drilled through the otic capsule. In most instances the observations were made after thin glass was laid over the opening in the cochlea. The observations in the inner ear were made on cats. Sound was conducted into the outer ear canal. The eardrum and ossicular chain were intact. Both the scala tympani and the scala vestibuli were opened.

The following observations were made:

1) With microscopic-stroboscopic methods, definite acoustic oscillations can be seen and photographed in both scalae.

2) The oscillation in the scala tympani is of opposite phase to the vibration in the scala vestibuli.

3) A strip of the cochlear partition executes vibrations between the two scalae.

4) There is a slight, but definite phase lag between the vibration of the cochlear partition and the eardrum. This phase difference indicates the travel time of the sound between the eardrum and the endpoint in the cochlea.

SUMMARY

Experiments on sound conduction in the ear were performed on living animals and on fresh temporal cadaver bones. The sound stimulus was applied through a tube into the external auditory meatus. The structures of the middle ear and of the inner ear were observed and recorded optically on motion picture film during acoustic stimu-

lation. The eardrum, ossicular chain, oval and round window, intracochlear fluid and the cochlear partition were studied as to their relative amplitudes and as to their phase relation. In living animals it can be seen that the stapes vibrates with a slight phase difference from the eardrum around an axis vertical to the footplate. There is a gliding side motion in the incudo-stapedial articulation whenever the stapedius muscle contracts. In man, a second type of stapes vibration around an axis parallel to the stapes footplate was observed (Bekesy). The air of the tympanic cavity which is frequently neglected by the students of sound conduction was studied. Experiments were performed in which the vibrations of the tympanic air were visulized by indicators. Small cotton fibers and microscopic smoke particles were used as tracers. These experiments prove the feasibility of mechanical indicators for air vibrations. The results obtained on specimens with intact eardrums and specimens with experimental perforations will be described in a different publication.

In rabbits the tensor tympani muscle contracts regularly in response to overthreshold stimuli. The muscle contraction is tetanic. Both muscles contract simultaneously. Under proper magnification, one can see acoustic vibrations of the muscle belly during strong contractions.

The structures of the inner ear were observed through "windows" in the otic capsule. In some experiments the windows were closed by thin glass and sealed with vaseline. It can be seen that the fluid in the two scalae vibrate in opposite phase. Secondly, a place of maximum stimulation can be seen on the cochlear partition. The vibrations of the cochlear partition have a definite phase lag with the eardrum vibrations.

950 E. 59TH ST.

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LXVIII

THE TECTORIAL MEMBRANE IN THE THEORY OF HEARING

THE SIGNIFICANCE OF THE INSERTION OF THE TECTORIAL MEMBRANE IN THE TRANSMISSION OF SOUND VIBRATIONS TO THE HAIR CELLS AND A THEORY OF THE MECHANISM OF TONE LOCATION IN THE COCHLEA

A. C. Hilding, M.D.

Duluth, Minn.

Your president has invited me to speak on some aspects of the theories of hearing with special reference to my own work. What I have to say will consist partly of observed fact and partly of theory. I shall deal especially with five aspects of the mechanical phases of cochlear activity. These five are: (1) the insertions of the tectorial membrane; (2) an anatomic arch recently described; (3) the translation of sound vibrations in the perilymph to stimuli of the hair cells; (4) the triangle of stimulus and its relation to intensity and (5) the location of frequency reception in the coil.

Most students of hearing for a long time have felt that the tectorial membrane in some way entered into the stimulation of the hair cells. It has long been suspected that the tectorial membrane was inserted in some way upon the organ of Corti but, until recently, these insertions had not been demonstrated.[6] In my studies last year at Columbia University, I worked out what I believe to be the approximate arrangement of these insertions. Briefly they are as follows: The outer margin of the tectorial membrane is inserted by a homogeneous net or membranous sheet upon the cells of Hensen. This net seems to extend over the entire convexity of the cells of Hensen down to the cells of Claudius on the basement membrane (and possibly even over the cells of Claudius). The insertion is not a simple one and involves more than one layer; it has previously been

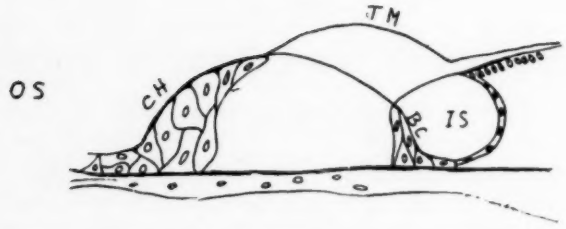

Fig. 1.—Diagrammatic representation of the two arches into which the tectorial membrane is incorporated. The larger one consists of the articulating zone of the tectorial membrane as the chief member supported by two abutments composed of the cells of Hensen on the outside and the border cells on the inside. These two cell masses are composed of loose cells and contain few if any fibers such as those found in the arch of Held. These two cell masses are mobile like shock absorbers, yield to the diagonal stimulating vibratory pull of the fibrils in the tectorial membrane permitting this to be transmitted to the hair cells. The other arch consists of the spanning zone of the tectorial membrane together with the border cells and the limbus. This arc spans the inner sulcus and forms it into a closed tube. This spanning portion of the tectorial membrane is yielding and flexible transversely and absorbs the up and down movements of the organ of Corti by an accordian-like action, thus preventing any stimulating motion between the tectorial membrane and the hair cells from up and down movement.

described in detail.[6] There is another line of insertions at Hensen's stripe, which is attached to the border cells in the internal sulcus (Fig. 2). This insertion seems to be similar in every way to the one at the outer border. The tectorial membrane, together with the cells of Hensen and the border cells, form an arch, which fits closely over the fibrous supporting arch described by Held (Fig. 1). These two cell masses form the abutments of the arch and support and maintain in position the main member, which is the articulating zone of the tectorial membrane. This articulating zone is the zone which overlies and is in contact with the hair cells. There is a second arch which bridges the internal sulcus; this is composed of the internal border cells, the limbus and the spanning zone (spans the internal sulcus) of the tectorial membrane. Between the cells of Hensen and the border cells lie the hair cells; they are at least in contact with the tectorial membrane and in all probability are actually attached. The obliquely placed fibrils, which are so apparent in the tectorial mem-

brane, have been thought by some to be long hairs from the hair cells,[1, 4] and I am inclined to agree.

In this case then, the hairs of the hair cells are very long in comparison with the size of the cells. The uppermost fibrils in the tectorial membrane would then belong to the outer row of the outer hair cells. The hairs of the next row of outer hair cells would lie immediately beneath those of the outermost row. Those of the inner hair cells would lie deepest of all. The fibrils are not all of the same length, some seem to end at the lip of the limbus, while others extend part way across the limbus. The longest extend some two-thirds of the way across the limbus and reach almost to the anchor bar of the zone of insertion of the tectorial membrane, i.e. these longest ones extend almost to the lamina ossea spiralis. To which cells these longest fibrils belong is uncertain.

Be that as it may, the tectorial membrane is supplied by numerous obliquely placed fibrils or hairs which extend from the limbus outward and toward the apex at an angle of about 20° or 30° with the tangent. If one regards them as hairs belonging to the hair cells, then they extend from the hair cell inward and toward the base instead of outward and toward the apex.

With this picture of the attached tectorial membrane in mind, I did some experiments designed to demonstrate the action of the tectorial membrane during rising and falling movements of the basilar membrane and organ of Corti. It was found in certain of the specimens studied that in up and down movements of the basilar membrane, the organ of Corti described an arc, the center of rotation of which lay about in the floor of the internal sulcus. The tectorial membrane arises at a point on the limbus which lies considerably higher, yet there was no shearing movement, as one could expect, between the articulating zone of the tectorial membrane and the hair cells. The arch just described rode pick-a-back upon the arch of Held and the shearing motion did not take place because the spanning zone of the tectorial membrane absorbed that movement by an accordian-like action. It has been found by a number of workers that the tectorial membrane is quite flexible transversely but is less flexible axially. In fact, axially it is rather non-yielding and somewhat stiff. This finding that there is no movement between the hair cells and the tectorial membrane during up and down movement would explain the fact that a large range of the basilar membrane

and organ of Corti may vibrate up and down with a single tone and yet not stimulate the hair cells excepting in one small spot. The search for the mechanism which localizes the stimulation has been long and arduous. Knowing the attachments of the tectorial membrane, one possibility now becomes quite apparent:

In up and down movement, the shearing forces, which are neutralized by the spanning portion of the tectorial membrane, act radially toward the modiolus and cause no pull upon the fibrils. If, however, there is a wave or bend with a slope directed toward the apex, there would inevitably be one zone where the forces of shear would act parallel to the direction of the fibrils. If the fibrils are non-yielding, as they seem to be from a number of points in evidence, then a pull would be made upon the hair cells, where the fibrils are attached.[7]

The deformation of the basilar membrane, as I conceive it, (considering now the negative phase) would be a very long slope directed toward the base and a sharper, shorter slope directed toward the apex. It would be on this latter that the stimulus would occur as the fibrils were tightened across the crest. To illustrate more graphically—picture a fisherman in a boat close to the shoreline. He makes a cast far up the shore and gets his hook snagged right close to the water line. The fisherman represents a hair cell and the pole and line represent the hairs or corresponding fibrils running through the tectorial membrane to the limbus. The shore represents the lamina ossea spiralis. If the waves upon which the fisherman's boat rises and falls are moving in toward the shore at right angles, as he stands holding his line, there would be little or no tension upon the hook from the waves. If, on the other hand, the waves are running parallel with the shore, then, as his boat rises on the slope of the wave away from his hook, there would be a tug on the line that would bend the pole and, as the wave passes under the boat and the boat slides down the slope directed toward the hook, the line would go slack. This represents the essence of my concept. The comparison, however, is not a true one because if the wave in the cochlea were a traveling wave, as described, then my concept would be fallacious. If there were a whole line of fishermen lying parallel with the shore, each one of whom had caught a hook in the manner described, then a wave traveling along parallel with the shore would pull the line of each one in turn. Such successive stimuli obviously do not take place in the cochlea. For that reason it seems to me that the stimulating wave cannot be a traveling one.

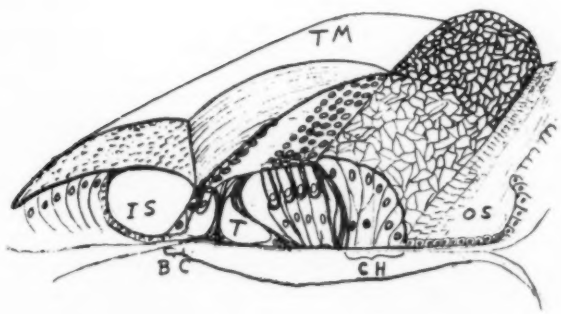

Fig. 2.—Diagrammatic representation of the insertions of the tectorial membrane (T.M.) upon the organ of Corti. I.S.—internal sulcus, O. S. —external sulcus, T—tunnel between the inner and outer rods, CH cells of Hensen, B.C.—border cells. A portion of the tectorial membrane has been cut away parallel with the fibrils exposing to view the one row of inner hair cells and four rows of outer hair cells. The fibrils are presumably hairs from the hair cells although the connection is not shown. The observer is looking toward the apex of the cochlea.

The outer border of the tectorial membrane comes to a feather edge at the outer row of outer hair cells and is attached to the upper surface of the cells of Hensen by a net or membranous sheet as indicated by the heavier lines outlining the ends of Hensen's cells (at the far end in the diagram). The under surface of the tectorial membrane is attached to the border cells along Hensen's stripe which lies at or just inside the row of inner hair cells. The under surface of the tectorial membrane between these two lines of insertion is the "articulating zone" which overlies the hair cells from which it is separated by a narrow space. The hairs supposedly span the space to enter the under surface of the tectorial membrane.

To summarize briefly thus far: The hair cells, which are supported in the "stutzbogen" or arch of Held, are conceived to have long fibrils which pass diagonally inward and toward the base into the tectorial membrane. The latter is supported and maintained in position over the hair cells by means of two cell masses which, together with this portion of the tectorial membrane, form an arch which is mobile. In up and down movement, this arch rises and falls with the arch of Held without any relative motion between them. It is only when the basilar membrane, the organ of Corti and the tectorial membrane are bent transversely, making a slope directed toward the apex, that a stimulus occurs. The stimulus occurs because the hairs or fibrils in the tectorial membrane, which are conceived to

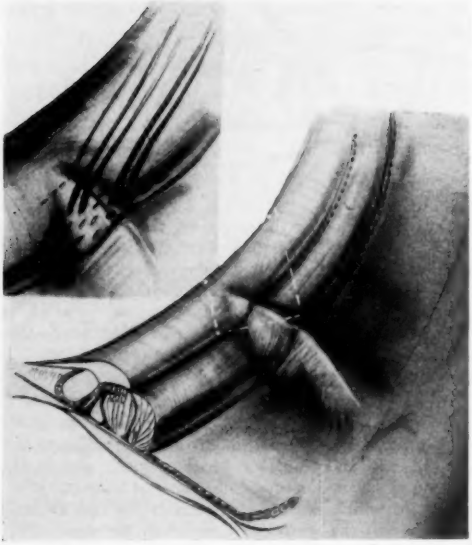

Fig. 3.—Illustration of a small portion of the contents of the cochlear duct looking diagonally downward upon the organ of Corti and the tectorial membrane. The proximal portion is directed toward the apex. The deformation, as conceived, of the basilar membrane and the contents of the cochlear canal taking place during sound vibration is depicted in the stimulating negative phase. There is a long, gentle slope extending from the crest of the deformation all the way to the base. It is upon the shorter slope facing the apex where stimulation is thought to occur. Only the inner portion of this slope which invades the region of hair cells would be effective. The triangular effective area is shown in the insert. The stimulus is conceived as taking place when the fibrils (or hairs) of the hair cells within this triangle are stretched across the crest of the slope.

be non-yielding, are put upon the stretch across the crest of this bend or wave.

Figure 3 illustrates the slope directed toward the apex approximately as conceived. It will be noted that it is in the shape of an ellipse. Only that end of the ellipse which invades the tectorial membrane and zone of hair cells would be effective in causing stimuli of the hair cells. It is illustrated as a small triangle (insert Fig. 3). If the slope does not invade this zone, the vibrations would supposedly be subthreshold. If only the apex reaches into this zone, it might include only a single cell in the outermost row of outer hair cells which would be stimulated. This would presumably be threshold stimulation. As intensity increases, this effective triangle invades deeper into the zone of hair cells and widens axially, including more and more cells. If it reaches to and includes the inner hair cells, maximum loudness is attained. If this concept is a true one, then one would expect the outer hair cells, which are stimulated oftenest and move through the widest amplitude, to be the first to show damage from overuse or acoustic trauma. Such is actually the case. The outermost hair cells are the first to degenerate.

LOCATION OF FREQUENCY RECEPTION

There is much evidence to indicate that each sound frequency has its own specific point of reception in the cochlear coil.[10] There have been many possible mechanisms suggested, none of which seem to explain this localization of frequency.

A mechanism which would explain placement of frequencies in the coil must fulfill certain requirements: For any given frequency, the mass would have to be constant. The stiffness, friction and inertia would all have to be constant. The response would always have to be the same no matter by what route the vibrations were introduced—through the oval window, the round window, an artificial fenestrum or through the bone. The response would always have to be the same no matter what the amplitude (within limits—there is a little shift in pitch with increased loudness). The response would have to be the same no matter what the phase might chance to be. It would have to be some mechanism so finely adjusted that it would permit of the recognition of 8,000 to 10,000 different tones. Among the mechanisms which have been suggested are the rods, the basilar membrane fibers and the tectorial membrane, the "tuned" hairs of

the hair cells, combinations of wave patterns and combinations of mass, stiffness and damping, the double fluid column between the round and oval windows and so on. None of the suggested mechanisms has been generally accepted.

During my dissections of the cochlea last year, attempts were made to study the movements of the basilar membrane and tectorial membrane by manipulating the fluid in the scala tympani at a rate of some four or five impulses per second. One of my first attempts succeeded beautifully. It was an isolated success. Every one of numerous specimens after that refused to show any movement in the basilar membrane. Eventually the now obvious reason occurred to me and the only strange thing was that the one isolated specimen did show movement. What occurred in all these specimens was that the fluid simply flowed back and forth through the helicotrema without moving the basilar membrane. In order to move the basilar membrane at such low frequencies it was necessary to partially cork the helicotrema, causing an impedance there greater than that offered by the basilar membrane.

The basilar membrane does not move at frequencies less than 25 cps.[2, 8] If the frequencies increase to 25, movement of the basilar membrane begins. As frequency increases, the apical portion comes to rest. The higher the frequency, the longer the portion which remains at rest and the closer to the base is the area of maximum vibration. This phenomenon is well known. The cause of it seems to be obscure and herein must lie the key to frequency placement.

Perhaps we have had our attention too closely focused upon the stapes and the basal turn as the source of activity. Perhaps we have been looking at the mechanism backward and the real activity begins at the apex and should be reckoned from there.

As the perilymph moves in response to sound vibration, it offers or encounters certain factors of resistance—friction, mass, inertia, viscosity—which might all be combined under the term impedance. The basilar membrane also offers certain impedance due to mass, stiffness, friction and tension. The stiffness according to Békésy[3] is graduated, increasing in the proportion of 1:100 from the apex to the base. At low frequencies, below 15, the impedance offered by the friction, inertia and viscosity of the perilymph is lower than that offered by any part of the basilar mem-

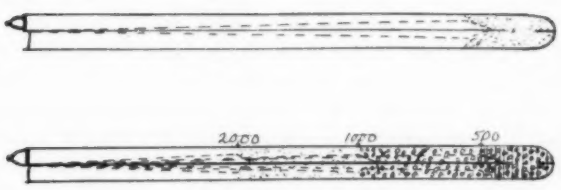

Fig. 4.—Diagrammatic representation of the cochlea unrolled, designed to show the clothes-pin action of the apical fluid column. The solid line (upper figure) indicates the basilar membrane in profile. The dotted lines indicate the excursion of the latter in response to sound vibration. The column of fluid in the apex which does not respond is indicated by stippling. Because of its inertia and friction, this part of the fluid cannot respond to the frequency in question and it "corks" the helicotrema. Its impedance (mass, inertia, friction, viscosity) at this frequency is greater than that of the basilar membrane so the basilar membrane yields to the pressure of the impulses. The degree of yielding along the basilar membrane depends upon the varying stiffness and is greatest at the point closest to the apex. At the point of transition between the vibrating and stationary portions of the basilar membrane, a slope is formed in the negative phase which is directed toward the apex. The hair cells on this slope are stimulated by the hairs or fibrils which are put on tension across the crest of this slope.

The lower figure illustrates the action of the cochlear fluid when three tones are sounded simultaneously. For each tone there is a given length of apical fluid which fails to vibrate and snubs the basilar membrane to a standstill at the indicated point as far as that frequency is concerned. For lower frequencies, the basilar membrane continues to vibrate at this point. Analysis could take place in this manner.

brane and the whole 70 mm flows smoothly back and forth through the helicotrema, without moving the basilar membrane. Time is an important factor. The force is applied slowly enough and long enough to move the entire double column. At 10 cps, for instance, the force would be applied for approximately 25 milliseconds. If frequency is increased to 500, the velocity of the moving fluid must increase. Every particle of the moving fluid must start from standstill, accelerate to a point, decelerate, stop, reverse, accelerate, decelerate and stop again 500 times a second. As the particle velocity is thus necessarily increased, the resistance to flow goes up greatly—approximately as the square of the velocity or of that order of magnitude. Meanwhile the duration of application of the moving force would be reduced to 0.5 milliseconds. The fluid at the apex in the helicotrema at 500 cps offers an impedance which is greater than

that of the basilar membrane. The inertia of all of the fluid cannot be overcome in such a brief space of time. Consequently, the basilar membrane yields and the vibrations short-cut through into the scala tympani, setting the basilar membrane into vibration, while the apical fluid remains at rest. The entire basilar membrane moves together in phase (or nearly so) excepting that apical portion which lies within the stationary apical fluid column—double column (Fig. 4). This portion is stopped as though a clothes-pin were slipped over it. The moving part of the basilar membrane moves with the greatest amplitude at the weakest point where the fibers are longest. The amplitude becomes progressively less as the base is approached but, if the gradation of stiffness increases evenly, there is some movement all the way to the base. It is as though the entire basilar membrane were swinging on a hinge pivoted at the base. The apical column, which can no longer follow along at the higher frequency, remains at rest and holds a corresponding part of the basilar membrane at rest. As the frequency is stepped up to 1000, there is a longer column of fluid at the apex, which can no longer follow along. The time now is reduced to 0.25 milliseconds. The impedance becomes still greater, the inertia cannot be overcome and exceeds that of the basilar membrane at a point farther from the helicotrema where the stiffness is greater and here is the point of greatest break through. The greater the frequency of vibration, the briefer is the time for overcoming inertia and the greater the impedance offered by this double apical column, the longer the stationary column grows and the farther the clothes-pin action extends toward the base. It is here that the clothes-pin action of the apical fluid column stops the up-and-down movement of the basilar membrane that a slope is formed which, in the negative phase of the movement, is directed toward the apex. It is here that the fibrils in the tectorial membrane, which are presumably hairs of the hair cells, are put on the stretch.

Here we have a mechanism which does not involve resonance (at least as we ordinarily understand it) and would fulfill the requirements listed above. It is a system of forced vibration. For any given frequency, the mass of the non-vibrating apical fluid would remain constant, as would the stiffness, friction, shape and viscosity of this non-vibrating portion. No matter where the impulses enter to set up the vibrations, whether they come through the round window, a fenestra or the oval window, a given length of column of fluid would remain stationary in the apex for a given frequency. If

the bone were vibrated about the fluid, as in bone conduction, the result would be identical.

Up to this point, we have not mentioned amplitude. An increase in amplitude would also call for an increase in particle velocity and further increase of impedance in the apical fluid. Would this not tend to shift the location and increase the pitch? It seemed to me that it would until Dr. E. L. Hill of the University of Minnesota, Department of Physics,[5] pointed out that it is not the total impedance which determines the point of stoppage but the difference in pressure on the two sides of the membrane and that the difference in pressure at a given frequency would remain the same regardless of the amplitude. He thought there might be a trifling shift one way or the other with changes in amplitude but, to use his words, "such a system would not be sensitive to amplitude."

This double column of apical fluid, which cannot follow the higher frequencies because of the increased impedance which it offers to the increased particle velocity, could well be the key to the location of frequency reception. This could be the mechanism that produces the slope directed toward the apex that is necessary to stimulate the hairs of the hair cells. The gradations could be so fine as to be infinitesimal and the number of possible tones infinite.

After reaching this point in my study a short article by Roaf[9] came to my attention. It describes this impedance of the apical fluid and the physical principles involved and was written thirty years ago. Stevens and Davis[11] some ten years later quoted Roaf's work but, in the same article, discussed the traveling wave concept, which they seemed to accept. These two concepts, to me, appear to be incompatible. I have seen no other references to Roaf's article.

SUMMARY

It was worked out on fresh specimens that the tectorial membrane is inserted upon the border cells in the inner sulcus along Hensen's stripe and that the outer border is inserted upon the cells of Hensen. The portion of the tectorial membrane between these two cell masses is attached to the hair cells (or is at least in contact). The tectorial membrane contains diagonal fibers, the exact nature of which is uncertain, but they are probably long hairs belonging to the hair cells. The tectorial membrane is quite yielding and flexible transversely but in the axis of the fibrils it is comparatively non-

yielding. The up and down movements of the basilar membrane do not cause stimulating movements between the tectorial membrane and the hair cells. This up and down movement is neutralized in the spanning zone of the tectorial membrane. Theoretically, however, when a bend occurs across the axis of the tectorial membrane and the organ of Corti, thus forming a slope directed toward the apex, the fibrils in the tectorial membrane are put on tension across this bend and stimulate the hair cells on the slope. The stimulated area is in the form of a triangle the apex of which is directed toward the modiolus.

A concept for frequency location within the cochlear coil, based on the same principles as those expressed by Roaf, is described. It pictures the basilar membrane as moving up and down as though hinged at the base for its full length from the base to the point of tone reception. Beyond this point it is held stationary by a double apical column of fluid which cannot vibrate at the given frequency. This double apical fluid column acts like a clothes-pin to hold the apical portion of the basilar membrane stationary. The point at which the transition from moving to stationary takes place is determined by the impedance of the apical fluid as compared with the impedance of the basilar membrane and the time during which the force of the impulse acts upon the mass of the perilymph.

626 Medical Arts Bldg.

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LXIX

TISSUE CULTURE TECHNIQUES FOR THE STUDY OF THE ISOLATED OTIC VESICLE

MERLE LAWRENCE, PH.D.

AND

DONALD J. MERCHANT, PH.D.

ANN ARBOR, MICH.

There is no greater single factor that can account so for our ignorance of the normal and abnormal metabolism of the inner ear than the inaccessibility of its structures. Imbedded in ivory-like bone they cannot be photographed like the retina nor can they be removed without grave damage. Even the histological preparation of the sensory cells and membranes is hampered by this dense, protective bone. If it were possible to obtain the membranous labyrinth free from its bony capsule, research on the biochemistry of the sensory elements could proceed unencumbered by the existing inaccessibility. This paper reports the attempts we have made to accomplish this by resorting to tissue culture techniques.

Our attempts to maintain in a culture medium excised portions of the organ of Corti or of the cristae of the semicircular canals from both the chicken and the guinea pig have proven unsuccessful so we have turned to embryonic tissue in the hope that the sensory elements could be made to differentiate and remain viable for further study.

The immediate goals of this endeavor have been to develop a technique whereby the tissue could develop completely undisturbed by either the feeding process or the manipulation on the microscope stage, to promote principally the growth of the cochlear area and to have this development obvious enough that dissection of the living sensory elements would be possible.

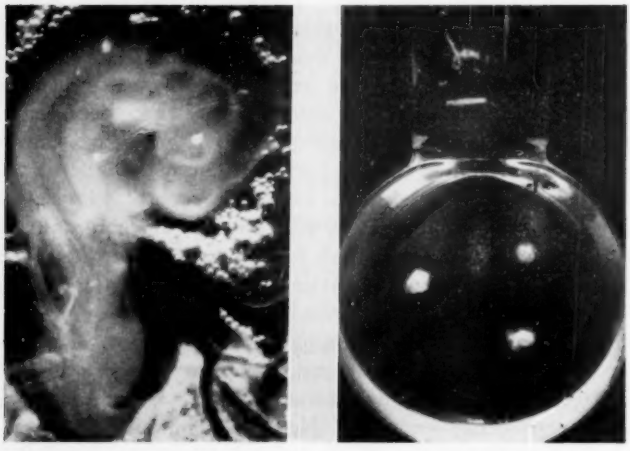
From the Department of Otolaryngology, the Institute of Industrial Health and the Department of Bacteriology, University of Michigan Medical School.

There have been no earlier attempts made specifically to grow the labyrinth in such a way that it could be studied in the living state. Investigators of embryological development have been interested in determining whether the otic vesicle will continue to differentiate independent of supporting structures, nervous connections or a vascular system. Hoadley[1] in 1924 reported the independent differentiation of the auditory vesicle removed from a three-day chick embryo and transplanted to the chorioallantoic membrane of a nine-day old chick embryo as host. In 1928 Fell[2] reported similar results, allowing the vesicle to develop on a medium of chicken plasma clotted with embryo extract. In both cases the degree of differentiation was checked by histological examination of the sectioned vesicles. Hoadley observed that, as the vesicle developed in the graft, there was much alteration in the relationship between parts of the membranous labyrinth and the surrounding mesenchyme, and Fell reported that the gross anatomical differentiation was slight, the original vesicular form being retained. Neither technique, however, permitted constant observation of the gross development and Fell's procedure, every 48 hours, of transferring the vesicle by means of a fine pipette to a fresh medium tended to keep the developing structure in the form of a sphere.

To be practical for the study of the growing vesicle the technique should provide for constant low-power microscopic examination. It should also permit daily patching and exchange of medium with minimum risk of contamination. And the procedure should produce no mechanical distortion of the developing tissue while at the same time holding it in place and yet allowing it to be removed for dissection or histological embedding. There are, of course, innumberable methods now employed in tissue culture studies, and it is difficult to predict in advance how the tissue of interest will develop or differentiate when grown by any particular method. Throughout the course of this study many different methods and media have been tried but there are many more that might be tried and may in the future progress of this work prove more successful.

METHOD

In the course of development of the embryo the first appearance of future auditory structures takes place in the mid-region of the myelencephalon as a thickened plate of ectodermal cells. This layer proceeds to invaginate: first to form a pit, then a sac attached to the



A B

Fig. 1.—A. Three day old chick embryo showing outline of otic vesicle at apex of neck flexure. 15X. B. Porter flask showing three vesicles in the culture medium. 1 1/3X.

surface layer by a hollow tube of cells called the placodal stalk.[3] Shortly this stalk detaches and the auditory vesicle briefly exists as a sphere of ectodermal cells with a slight protuberance on one region (dorsal) of its surface. Figure 1(A) is a photograph of a living three day old chick embryo showing the otic vesicle as a white outline at the apex of the neck flexure. From this vesicle develops the entire membranous labyrinth and if caught at the stage shown in the photograph it can be cleanly removed from the rest of the embryo.

Close observation of the photograph reveals a whitish mass just anterior to the vesicle. This is the acoustico-facialis ganglion, the acoustic portion of which will send fibers into each of the developing sensory areas. Since it has been reported that this nervous tissue is not essential to the normal histological differentiation of the vesicle we have been careful to remove it along with the surrounding mesenchyme in order to have the vesicle as clean as possible. However, it should be noted that the acoustic ganglion, which gives rise to the

eighth nerve, originates from the anterio-medial wall of the vesicle itself[4] and remains so intimately connected with this wall that it is doubtful whether the ganglion can be removed without doing some damage to the otic sac. In the removal of our most recent vesicles we have left the ganglion attached and have observed some interesting new developments. These, however, do not alter the observations recorded here and so will not now be reported.

Removal of the vesicle is carried out under aseptic conditions and in Hank's balanced salt solution. Generally both vesicles are removed from a number of embryos at one time. Once removed and free from extraneous tissue they are ready to be placed in the culture medium.

Before transfer of the vesicle the medium is prepared in the form of a clot in a suitable container. For the purposes of these experiments the Porter flask has proved to be the most convenient and satisfactory. It consists of a tube 15 mm in diameter blown out at the end so as to form a bulb 35 mm in diameter flattened on two sides in order to provide undistorting optical surfaces for microscopic work. The neck of this flask can, of course, be of any length, but for use on a microscope stage the short neck is preferred since it is easier to maintain the flask in position. Figure 1 (B) shows this flask with three growing vesicles in the clotted medium.

For the growth of the chick vesicle the most successful medium has been a clot formed of equal parts of chicken plasma and chick embryo extract. Six drops of plasma are first placed in the sterile flask and spread around with a pipette so the liquid covers the entire area of one of the flat surfaces. Six drops of the embryo extract are then introduced and spread throughout the plasma. A clot will generally form in less than a minute. The vesicles are then carried by means of a fine pipette from the balanced salt solution to the flask where they are gently placed on the surface of the clot, generally two to four to a flask. The flask is then closed with a rubber stopper and incubated at a temperature maintained, as nearly as possible, at 38° C. Within six hours some of the mesenchyme cells which surround the vesicle and unavoidably are removed with it show division and growth as the cell mass spreads out into the clot.

The greatest hazard to the success of this procedure is liquefaction of the clot. If the plasma is not fresh this may occur within

24 hours. Any excess agitation of the clot will also cause it to liquefy. Even with very fresh plasma the clot will liquefy around the vesicles in some instances. When this happens the developing vesicle and its growing peripheral cells shrink into a tight ball and start to degenerate. The chances of this happening are much less with fresh plasma but it does happen and we have yet found no satisfactory method of preventing it. One possibility is to provide the vesicle with a solid meshwork embedded within the clot upon which the mesenchymal cells can build up providing an anchorage, so that if the clot should liquefy the vesicle will retain its shape until a fresh clot is provided. For such a meshwork we have tried fibrin foam, gel-foam and cellulose sponge. The first two of these have such fine pores that microscopic observation of the vesicle is prevented. The small-pored cellulose sponge serves best. By cutting the sponge into very thin slices the pores appear as holes in this thin layer. If the vesicle is carefully placed in one of these small holes the peripheral cells grow out and over the sponge fiber boundaries thus serving to anchor the vesicle should the clot liquefy. For purposes of observing the earliest stages of differentiation this works quite well but the vesicle soon grows to cover an area larger than that of the hole in the sponge so microscopic observation is again prevented and nothing has been gained. For our purposes the most satisfactory procedure has been to use fresh plasma and plant a number of vesicles in several flasks, saving for study those which do not liquefy.

RESULTS

The Chick. To date we have explanted around 227 otic vesicles from chick embryos. Many of these have been expended in developing the technique, some have been carried as long as possible in order to observe the gross development, others have been sectioned for histological study and a few, while in the living state, have been dissected under the microscope.

As in vivo the first structure to become noticeable is the endolymphatic duct. In only one, of all the vesicles studied so far, did this actually grow out into a duct terminated by an endolymphatic sac. In all the others it appeared as an area of many convolutions. Histologically, however, this area appears as a normal duct appears at a comparable age.

In placing the vesicle in the plasma clot it is difficult to be consistent in the manner of orientation. For this reason it is not always

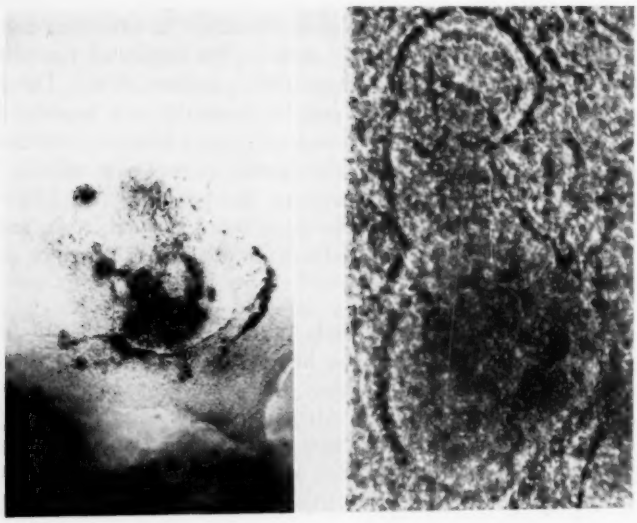
possible to predict where certain structures will appear, but after 24 hours in culture the formation of the endolymphatic duct serves as an indicator for the orientation of the vesicle. In vivo this duct appears as an apical (dorsal) outgrowth in the region of the placodal stalk and in fact carries the vestige of this stalk with it. The dorsal external wall of the vesicle expands to form the pars superior labyrinthi and in so doing shifts the endolymphatic duct medialward and forms an indistinct groove on the median side of the vesicle. This groove marks the separation between the pars superior labyrinthi, destined to form the semicircular canals and the utriculus, and the pars inferior labyrinthi which will develop into the sacculus, ductus cochlearis, and lagena.

In vitro the vesicle proceeds to develop normally up to this point and the three divisions can be recognized, but from this stage on, gross anatomical development seems to be somewhat arrested especially in the pars superior. Although incompletely formed canals are occasionally seen, in no instance, so far, have these been observed to develop completely. However, as will be shown shortly, the sensory areas apparently are differentiating normally.

The region of particular interest for our purposes is the pars inferior and its ductus cochlearis. The gross development of these parts is much more normal and one can see the cochlea extending away from the superior part of the labyrinth. In fact, this membranous portion is transparent enough so that some of the structures within the cochlear duct can be identified.

Figure 2(A) shows a vesicle which has developed an endolymphatic duct appearing in its typical (in vitro) convoluted form. The pars superior can be seen still retaining a vesicular form while to the left the cochlear duct and sacculus can be seen. At the time these pictures were taken this vesicle had been in culture a few hours over three days. In vivo at this same age lobes for the canals would have been formed. Close examination of the pictured vesicle shows the faint outline of the lateral canal on the lower left of the outer boundary of the pars superior.

Figure 2(B) is a high-power view of the pars inferior development and one can see the lagena, which will be described shortly, clearly outlined at the tip of the cochlea. The large round portion at the lower end attached to the pars superior is the sacculus. The

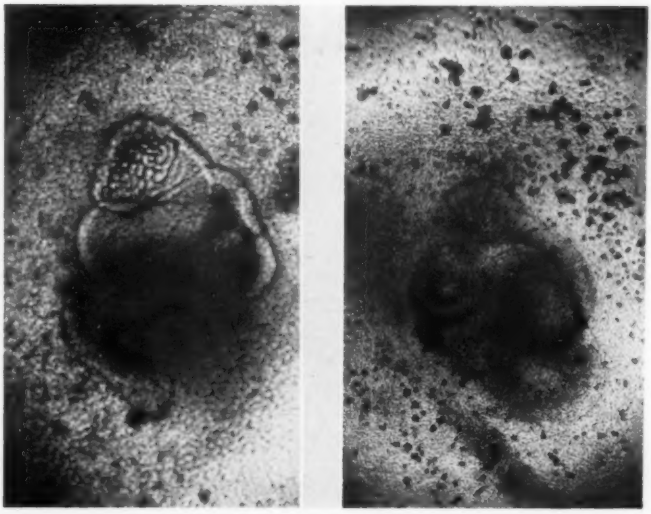


A B

Fig. 2.—A. Developing chick vesicle after three days in culture following removal from three day old chick embryo. The sacculus, cochlear duct and lagena appear as an outgrowth to the left of the vesicle. The convoluted area at the top is the endolymphatic duct. Living and unstained. Chick ves. no. 34. 23X. B. High power photomicrograph of sacculus, cochlear duct and lagena of vesicle shown in A. The lagena at the tip of the cochlea is clearly outlined at the top of the picture. Living and unstained. 247X.

relative size of this structure is rather abnormal for this embryonic age. In vivo the sacculus is not apparent until the seventh day when it appears only as a slight protuberance on that portion of the pars inferior nearest the pars superior.

Phylogenetically there is considerably more variation in the development of the pars inferior than the pars superior and one finds, in the adult bird, a cochlea that is a short, slender tube only slightly curved with a basilar membrane only 2.7 mm long while in man the same structure is long and coiled over two and three quarters turns on the average with a basilar membrane about 31.5 mm long.



A B

Fig. 3.—Developing chick vesicles three days in culture following removal from three day old chick embryos. Both show the typical separation at this stage into the three basic divisions: the endolymphatic duct appearing as a convoluted area at the top, the pars superior and the pars inferior. Living and unstained. Chick ves. nos. 218 and 214. 30X.

As can be seen from the magnification of the photomicrograph this cochlea, while assuming its normal form of development, still has considerable growing to do.

Figures 3A and 3B are pictures of other vesicles in culture three days. The typical appearance of the otic vesicle in a six-day chick embryo can be seen as the structure divides into its three distinct parts.

The dark areas in these pictures are the beginnings of cartilage. The mesenchyme which surrounds the otic vesicle develops into the perilymphatic system and the bony capsule which later surround the entire membranous labyrinth. It is impossible to remove the vesicle without some of these mesenchyme cells. One of the problems yet

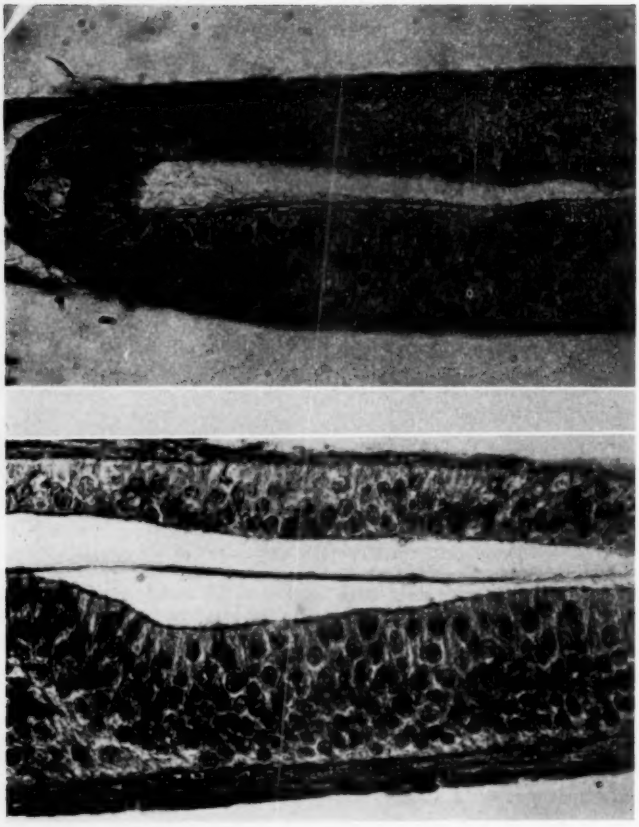


Fig. 4.—A. Photomicrograph of the lagena from a vesicle cultured 7 days after removal from a 3 day chick embryo. Fixed in Bouin's fluid. H and E stain. Chick ves. no. 6. 350X. B. Photomicrograph showing tectorial membrane and hair cells of the organ of Corti from the same vesicle as in (A). 450X.

to be solved in this work is the prevention of this cartilage formation which so far has prevented the further observation of the vesicle's gross development after three or four days in culture. The progressive differentiation continues, however, until the embryonic structure is 10 to 11 days old. At this age it has increased to such a size that the innermost cells can no longer obtain nourishment from the medium and degeneration starts. Once this happens the entire tissue rapidly disintegrates and the preparation is lost. That normal histological differentiation has taken place up to this point is demonstrated in Figures 4 and 5 which show various sensory areas in two vesicles fixed in Bouin's fluid after 7 days in culture.*

In the bird the terminal portion of the cochlear duct develops into a special structure called the lagena. It is a structure much like the other static organs of the labyrinth consisting of a layer of sensory cells topped by a membrane and otoliths. Figure 4A shows the embryonic form of this structure and for this stage it appears normal. The sensory cells and their "hairs" with the resting membrane can be clearly distinguished. The same is true of the sensory cells of the organ of Corti, more properly called the papilla basilaris in the bird since there is no arch of Corti with its pillar cells as in the mammal. This structure is shown in Figure 4B. The tectorial membrane extends from the left down over the sensory cells. These cells are just forming but can be clearly distinguished by their protruding "hairs" and their large nuclei. Note that this portion of the membranous labyrinth is surrounded only by a very thin layer of cells (the plasma clot has been removed) showing that up to this stage at least the cochlear duct has been maintained alive and apparently normal without a bony capsule.

There are eight sensory areas in the labyrinth of the bird: the papilla lagena, papilla basilaris, macula sacculi, macula utriculi, macula neglecta, and the three cristae of the ampullae of the semicircular canals. Of these all but the macula neglecta could be found in the vesicles being described. Some of the structures, particularly the cristae, were in very early stages of differentiation and it is possible that this rather insignificant structure, the macula neglecta, had not yet reached a stage where it could be recognized. To give a further

*We are indebted to Dr. Alexander Barry of the Department of Anatomy, and to Miss Doris M. Gardner of the Department of Bacteriology, University of Michigan Medical School for the preparation of these sections.

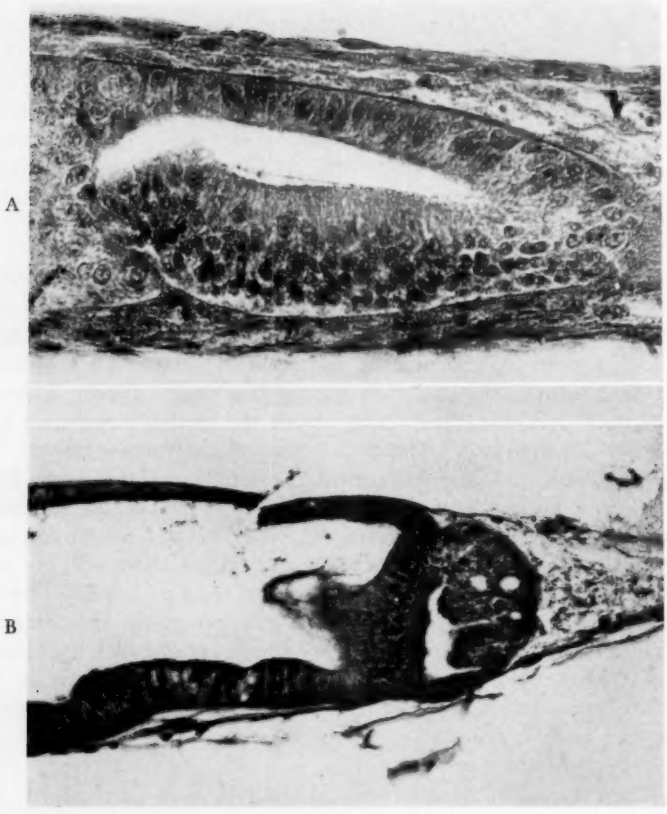


Fig. 5.—A. Photomicrograph showing the otolithic membrane and sensory cells of the macula of the utriculus taken from the same vesicle shown in Fig. 4A. 480X. B. Photomicrograph showing the cristae of an ampulla in a vesicle cultured 8 days after removal from a 3 day chick embryo. The convoluted structure to the right of the vesicle is the developing endolymphatic duct. Fixed in Bouin's fluid. H and E stain. Chick ves. no. 9. 240X.

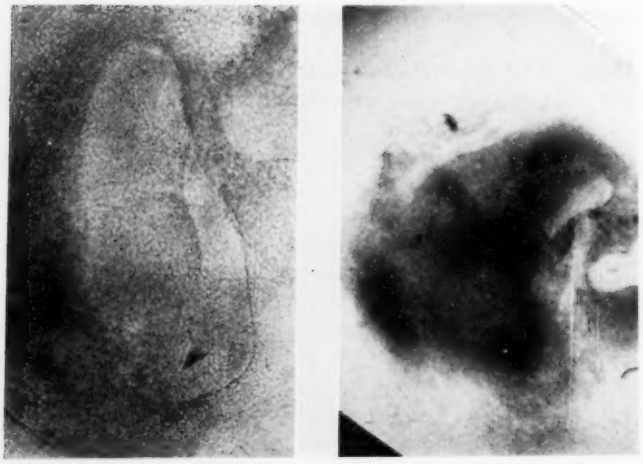
idea of how these areas have developed, Figure 5A shows a photomicrograph of the macula utriculi of this same vesicle.

Under these conditions of growth the rate of cell differentiation in the various sensory areas seems to differ for each vesicle. This is probably more the result of the effects of the culture process than of peculiar characteristics of the chick otic vesicle. As mentioned, seven of the eight sensory areas were apparent in the vesicle, three of which are shown in Figure 4 and 5A. In this labyrinth, however, the cristae had not yet formed a cupula and their structure was flat consisting of differentiating sensory cells covered by a thin membrane. In another vesicle of the same embryonic age, however, the cristae appear much more advanced and Figure 5B shows a picture of one of these. Study of the serial sections through this region shows that the cupula was developing quite normally. It is obvious that no canal has yet formed, while in vivo they are generally completely "pinched off" by the seventh day.

It is rather unreasonable to expect normal gross anatomical development in a membranous structure deprived of the external molding forces that must exist in a normally developing embryo. Such a deprivation should not, however, interfere with the development of sensory areas and this seems to be the case.

The Rat. Because of the accuracy of determining the age of the embryo and the ease with which it can be removed, the chick is ideal for these studies. But, as mentioned, the organ of Corti in the bird is quite dissimilar to that found in the mammal and since the main purpose of this study is to isolate the cochlear duct and its sensory cells it would be more appropriate to grow a mammalian vesicle if further experiments on these sensory cells are to apply to man.

For these studies we are now using the hamster, but our first attempts were on the white rat. The difficulties of such procedures on mammals are obvious, the main one being to determine the correct age for removal of the embryo. To solve this, several rats were bred at the same time. This is difficult to determine precisely but apparently an estimate within six hours is close enough. These animals were then opened on successive days and on the ninth day several vesicles were obtained and placed in the chicken plasma clot.



A B

Fig. 6.—A. Photomicrograph of rat vesicle 18 hours in culture after explant from 9 day old embryo. Living and unstained. Rat ves. no. 2. 110X. B. Same vesicle as in A, 68½ hours after explant. The vesicle is only faintly outlined because of the formation of cartilage, but the endolymphatic duct can be clearly seen at the top right of the vesicle. Living and unstained and pictured at a magnification much less than in A.

These experiments on the mammalian otic vesicle have just been started and are reported here to show the feasibility of using such material. On these first vesicles much extra mesenchyme was also removed with the result that the development of cartilage followed explanation quite rapidly. Several pictures of gross development of the specimen were obtained,* however, before it became obscured; two of these are shown in Figure 6. The first one, Figure 6A, was taken 18 hours after explant and bears a remarkably close resemblance to reconstructions that have been made of the human vesicle at a comparable age.[3] The endolymphatic duct is extending toward the top. The more darkly shaded spherical structure in the center is the vestibule and the cochlear portion is forming at the bottom.

*For these and many other photomicrographs taken but not shown we are indebted to Dr. Ruth Lofgren, Department of Bacteriology, University of Michigan Medical School.

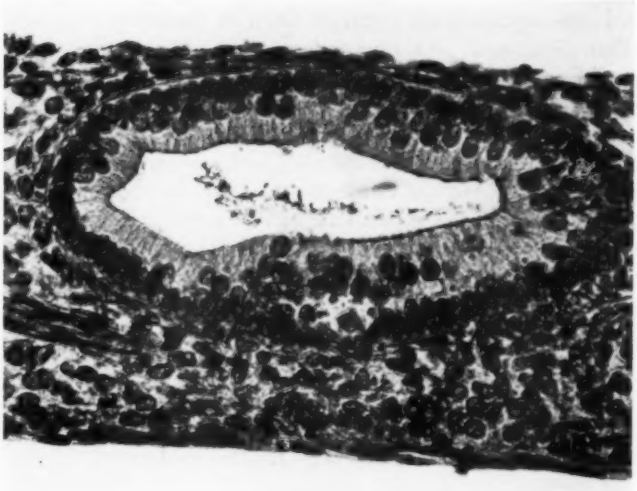


Fig. 7.—Photomicrograph of a portion of the cochlear duct of the rat vesicle shown in Fig. 6. Fixed in Bouin's fluid 8 days after explant and stained by Bodian's silver method. The tectorial membrane does not take this stain and so cannot be clearly identified but it arises from the cylindrical cells at the right of the duct and extends over to the region where the cells shorten and flatten at the lower left. This region will become the external sulcus and the three nuclei at the top of the rise are the developing external hair cells. Just to the right of the upper nucleus a rod can be seen forming and further careful scrutiny shows the invasion of nerve fibers into this region from outside the duct. The cells surrounding the duct are cartilage. Rat ves. no. 2. 460X.

Figure 6B shows this same vesicle 68½ hours after explant. Because of the density of the forming cartilage the actual structure is somewhat vague. The endolymphatic duct can be seen extending off to the right near the top of the vesicle and the rest of the form can just be made out as an expanded, oval structure.

This vesicle was kept in culture a total of eight days making its embryonic age actually 17 days. At this time it was fixed in Bouin's fluid and sectioned serially for histological study.*

*We are indebted to Dr. Kay T. Rogers and Mr. Gerald Abrams, Department of Anatomy, University of Michigan Medical School for the preparation of these sections.

No canals had developed but an uncoiled cochlear duct had formed and contained an organ of Corti in the early stage of differentiation. Figure 7 is a photomicrograph showing this embryonic organ of Corti. Unfortunately the fine strands of the tectorial membrane do not show but they arise from the long slender cells at the right and extend over to the region where the surface slopes down to the future external sulcus cells. The large nucleus at the beginning of this slope and the two beneath it are the outer hair cells and just to the right of the uppermost nucleus a rod can be seen forming with the inner hair cell to the right of it. The structure is obviously differentiating and presumably if kept a little longer would form a normal adult organ of Corti.

This specimen was stained by Bodian's silver method making visible the invasion of nerve fibers toward what we have just described as outer hair cells. Note again that although the extraneous tissue is thicker here than in the case of the chick, the cochlear duct is well isolated and surrounded only by this thin layer.

CONCLUSION

With these encouraging results at hand it appears that, in the use of the methods of tissue culture, a technique is available for the isolation of the sensory elements of the membranous labyrinth.

Further refinement of the technique should provide a means for carrying the development of the tissues into the adult form. It should be possible to reduce the density of cartilage formation and to eliminate the hazard of degeneration by reducing the size of the developing vesicle through dissection and further explanation of the areas of interest. Such dissection has already been successfully performed and the parts maintained in a viable state in the culture medium. These unstained tissues, however, present a completely unfamiliar picture under the microscope and appear quite different from the typical fixed and stained specimens.

From what we have seen of the cytological differentiation in the entire cultured vesicle it would seem unlikely that isolation and manipulation of the area cause the altered appearance. More likely the structure in the living condition has characteristics the nature of which will have to be determined before the material can be used for analytical study.

UNIVERSITY OF MICHIGAN MEDICAL SCHOOL.

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(Scientific papers of the American Otological Society will be continued in the December issue.)

The Scientific Papers of the American Broncho-Esophagological Association

LXX

THE PRESIDENT'S ADDRESS

FRED W. DIXON, M.D.

CLEVELAND, OHIO

It is a pleasant task to welcome you to the 34th Annual Meeting of the American Broncho-Esophagological Association. I wish to thank you for the privilege of serving as your President. It is a very great honor and one which I sincerely appreciate.

As we look down the path through the 36 years of growth of this Association we see many changes. At first, in 1917, called the American Asociation of Per-Oral Endoscopists, it finally, in 1939, became known as The American Broncho-Esophagological Association. There were, in 1917, thirty-two founder members of which today six are alive: Dr. Chevalier Jackson, Dr. Thomas Halsted, Dr. Harris Mosher, Dr. Ellen Patterson, Dr. George Coates and Dr. Fielding Lewis. We now have a combined membership of 243 of which 160 are active members.

At first the only requisite for membership was that the applicants were broncho-esophagologists or otolaryngologists who showed an interest in endoscopy. In 1934 it was voted that all applicants for membership must first be certified by the American Board of Otolaryngology. Finally, applicants certified by other Specialty Boards were admitted. Today 87% of our members are otolaryngologists, while the remainder are about equally divided between broncho-esophagologists and those specializing in diseases of the chest.

The program of the Association in the beginning dealt mainly with removal of foreign bodies, the instruments used, and the anatomy of the lungs. In 1926 several papers were presented on Lipiodol the "lung mapper." Finally the endoscopist demonstrated that many cancers in the chest occupied a localized, well defined area. Gradually the medical profession began to realize that the broncho-esophagologist

was pointing to the life saving operability of cancer of the lungs and esophagus.

Today most of the papers presented deal with chest diseases and their management, so what began as a narrow specialty, has now broadened into a diagnostic and treatment field affecting many specialties. Naturally, this results in overlapping and competition. In a hospital where diagnostic aid is sought, three things are of the essence; prompt service, a minimum of trauma, and a well considered opinion. The bronchologist should be familiar with the normal and abnormal findings and be able to evaluate these for the clinician. He should describe in detail any narrowing, fixation, inflammation, secretion or obstruction. Secretions and biopsies should be given to the pathologist immediately. Injections of opaque oil under fluoroscopic guidance is preferable. Any department furnishing this type of service will have a deserving place on the diagnostic team.

Out of 3,000 otolaryngologists certified by the American Board of Otolaryngology, only 138 are active members of this Association. The average age of the active members of this Association is 56 years. The future of any society is in its youth. Young men have the energy, the desire to succeed, and the capacity for leadership. Let us stimulate and guide their efforts. If we are to maintain a strong, vigorous Association, each of us must interest and sponsor one or two younger otolaryngologists in this field so that they may become eligible for membership in this Association. Today when many large otolaryngological practices are being abandoned for lack of properly trained otolaryngologists, the conclusion is reached that the specialists are shrinking faster than the specialty.

It is a noteworthy fact that the papers presented on our program in the past six years have been given in almost equal numbers by the otolaryngologists, the thoracic surgeons, and the bronchoesophagologists. The meetings have been well attended. This would indicate that our own members are aware of the importance of keeping abreast of the newer developments in this branch of our specialty and their need to have these developments brought to their attention, even to the extent of its being missionary.

It has been customary to decide at the January Council meeting where we will meet the following year. Due to the shortage in hotel facilities, I believe it would be advisable for our Council to decide upon out meeting place three years in advance.

The question of a combined meeting with the thoracic surgeons, the chest physicians, the American Medical Association, or

the otolaryngological societies, as we are now doing, has perplexed the Council each year. It is undeniable that the excellent programs offered by these various Societies are extremely worthwhile and their meetings could be profitably attended.

Today there is a public demand that the rugged isolationism of the medical profession be replaced by our leadership in civic affairs. Good public relations and our help in solving community problems are necessary if we are to avoid the curse of socialism. These obligations are time consuming and when added to the meetings of our hospital staff, our County Society, our State Society, our American Medical Asociation, our Academy, and our three Otolaryngological Societies, it causes us to question the advisability of adding another meeting to this list.

In November 1952 a questionnaire was sent to 156 of our active and associate members. Of this number 132, or 85%, were returned. In reply to the first question—"Do you favor the continuance of meeting with the American Laryngological, American Otological and the Triological Societies as we are now doing," 99, or 75% answered "Yes," 33, or 25%, replied "No." However, many said they would not object to an occasional meeting with another group.

In reply to the question as to what group they preferred, the American Association of Thoracic Surgeons and the American College of Chest Physicians led the list.

Your Council, meeting in January, were of the opinion that in deference to the minority group, an occasional meeting with another group might be considered.

Five years ago our Association was operating in the red. By increasing our dues, plus the efficient management of Dr. Clyde Heatly, our Treasurer, we now have a comfortable bank balance. While this is not large, it seems adequate for our needs. This money is invested in government bonds.

Three years ago a Committee was appointed to investigate the feasibility of publishing our Transactions and our own Journal biannually. While it was generally agreed that this would more firmly enhance our position, due to the ever increasing cost of paper, printing, and office personnel, it was decided our budget would not allow this increased expenditure.

I have already made a plea that you encourage younger men to join our ranks. To become eligible for membership, they must sub-

mit a thesis, or not less than five published articles reporting their own work in broncho-esophagology or the endoscopic phases of laryngology, gastro-enterology or thoracic disease. The fear of criticism, the lack of something new and startling, coupled with mental inertia, produces a barrier which disheartens the applicant. We can help him overcome this obstacle by encouragement, by furnishing him with some of our interesting findings, and by coaching him in his writing. While this will be a service to the Association, it will be a greater service to the individual, for it stimulates him to study and to share his knowledge which is so essential to medical progress.

Before starting any scientific writing, urge the writer to obtain some information on the art of writing. Probably it would help all of us to read: "Writing a Medical Paper—Why and How" by Wesson[5] "The Writing of Medical Papers" by Mellish[3] or "Medical Writings" by Jordan and Shepard,[2] or "The Art and Practice of Medical Writing" by Fishbein and Simmons,[1] and others.

In my capacity as Editor of the Transactions of this Association, many manuscripts have been outstanding. There was at their beginning a few sentences outlining the purpose of the paper, then followed the meaty framework with its clear summary and logical conclusions. In writing medical papers the following rules are helpful: Short sentences, clearly and accurately composed, make a forceful sentence. Simple words properly used are understood best. Eliminating useless words reduces the cost of publication and saves the reader's time. Prepare two papers, one for reading, which should be a condensed version to hold the attention of the audience, the second one for publication should give complete details. Photographs should be glossy prints 5x7 inch negatives. Uniformity of prints is helpful. Due to the cost of making the engravings, it has been the policy of this Association to pay for the first six prints, the author bearing the expense of additional ones. Color reproductions are expensive. An average color plate will cost as much as a fishing trip to Canada. In submitting an illustration, its number, the word "top" and the name of the author, should be lightly written with pencil on the back of each print. The legends of all illustrations should be typewritten on a separate sheet of paper, for the legends go to the printer, while the photographs go to the photo-engraver. Tables and charts are used for clarity and comparison. They should be brief, easily assimilated and should earn their place. They should be submitted on separate sheets of paper. Any pen sketches should be in India ink. A bibliography alphabetically arranged is desirable. Its length is not a criterion of the value of the manuscript.

In the preparation of a program, time should be allowed for discussion of a paper as the discussion is often the most valuable part of the program. When this discussion is sent to those participating, may I make a plea for its prompt return to the editor. Immediate return hastens completion of the Transactions and gives the early satisfaction of seeing that you have contributed something to making the meeting worthwhile.

In summary, examination shows our Association is structurally and financially sound. I believe our attention should be focused on securing young members who are interested in broncho-esophagology.

In closing, I wish to thank Dr. Edwin Broyles, our Secretary, who arranged this program, Dr. Clyde Heatly who has guarded our treasury, Dr. Francis Davison, our painstaking Editor, and the members of the Council for their helpful guidance while President of this Association.

1027 Rose Building.

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LXXI

EXPERIMENTAL TRACHEAL IMPLANTS

JOEL J. PRESSMAN, M.D.

LOS ANGELES, CALIF.

Resected sections of trachea involving its entire circumference have not been successfully reconstructed by anyone using solely the tissues of man or experimental animals.

True enough segments of tracheas, if not too long, have been resected and restitution of continuity established by mobilizing the remaining proximal and distal segments thus enabling one to bring the severed ends together; and portions of the circumference have been restored by myself and others through the medium of a patch of fascia lata or skin and other materials as well, but the entire circumference of the trachea has not been successfully re-established as a rigid enough tube without the use of foreign materials as supporting structures.

To one interested in surgical experimentation this presents a fascinating challenge which can be accepted with the hope of fair chance of success only by starting at the very beginning and learning all one can about the phenomenon of tracheal regeneration from every point of view. As a result of such experimentation and observation, several basic axioms have been established, and I shall cite and illustrate them to you.

Axiom 1: If a section of trachea is resected and replaced by any sort of mould, such as a polyethylene tube (Fig. 1) which is telescoped into each end, a new, complete fibrous tube (Fig. 2) will grow about the polyethylene and be adherent to the severed ends of the trachea. It will eventually be lined with epithelium, but it will not be rigid.

A successful clinical case involving such a procedure was carried out and reported by Dr. William P. Longmire, Jr. and formed the basis and inspiration for this work.[5]

From the Department of Surgery, School of Medicine, University of California at Los Angeles.

Supported by a research grant from the National Cancer Institute of the National Institutes of Health, U. S. Public Health Service. (No. CS-9241)

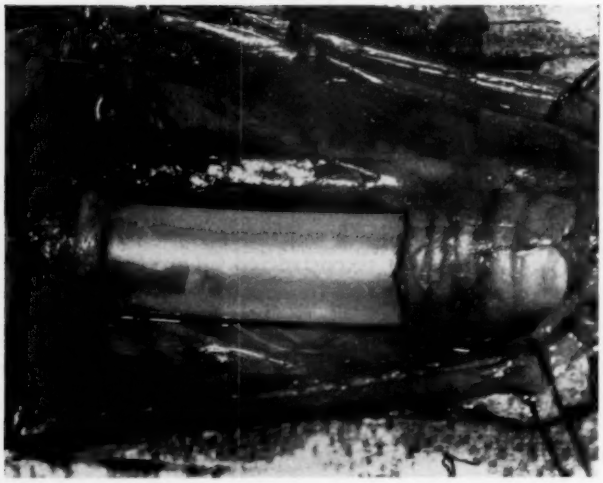

Fig. 1.—Polyethylene tube replacing resected segment of cervical trachea.

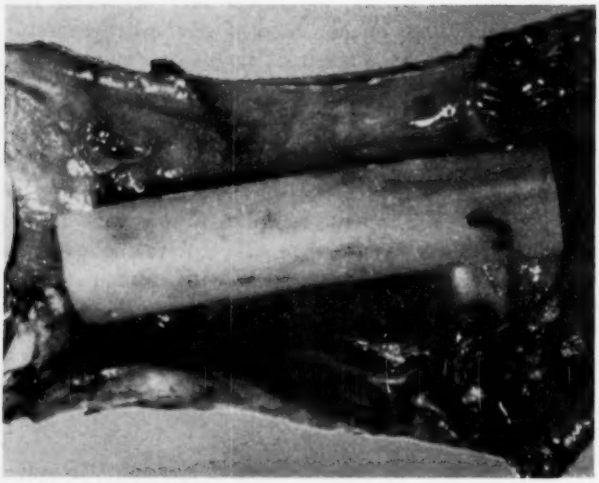

Fig. 2.—Epithelium-lined fibrous tube which has grown around polyethylene implant.

Fig. 3.—The segmental distribution of blood vessels to the trachea.

Axiom 2: If the polyethylene tube is allowed to remain in situ for as long as four months, it is very likely that granulomata will develop about the end of the tube and occlude the airway, or this may occur much earlier. The speed and degree of granuloma formation seems to vary directly with the movement of the polytheylene tube which traumatizes the mucosa, but this occurs even if movement is minimum as when the tube is snugly fitted and anchored to the trachea with tantalum sutures. The sutures between most polyethylene implants and the trachea will not hold due to tracheal movement, and the polyethylene will come to lie as a free foreign body. This enhances the growth of granulomata.

Axiom 3: If in an attempt to lend rigidity to the epithelium-lined tube thus formed, a sponge-like material such as Ivalon is wrapped around as a stiffener (in the hope that the polyethylene tube might subsequently be removed), fibrous tissue will grow into the meshes of the stiffener; but this will take place only on the external surface which is everywhere in contact with tissue. It will not grow on the inside but extends down from the severed ends of the trachea for only a few millimeters.

Axiom 4: In all experiments so far described, the end of the insert has been telescoped into the trachea. Our fourth axiom states

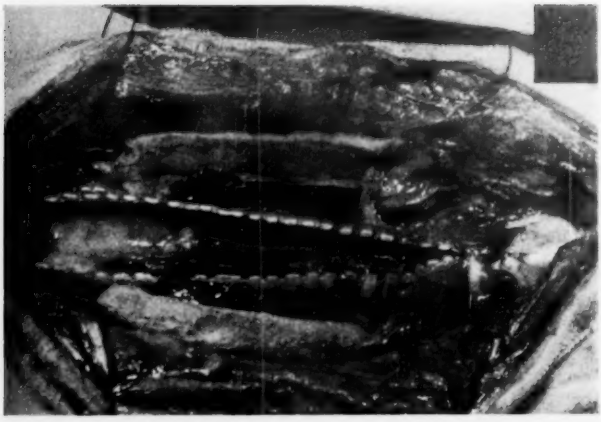

Fig. 4.—Necrosis of the trachea following isolation from segmental vessels.

that it is not possible to reverse this and telescope the tracheal stump into any tube or foreign substance since the segment so invaginated will necrose. This necrosis is due to the peculiarities of the blood supply of the tracheal rings which is segmental (Fig. 3), each pair of rings having an essentially independent supply of blood from vessels which branch off at intervals from a vessel running lengthwise with the trachea, the segmental vessels passing horizontally to the successive pairs of rings.

To further prove that not more than one or, at the most, two rings can be isolated from these transverse vessels by anything wrapped around them the following experiment was performed and not even one ring survived.

A sheet of tantalum foil was wrapped completely about a segment of trachea which had been isolated from its bed but not severed. A thin layer of polyethylene sponge was then wrapped about the foil, but the sponge was permitted to extend for a distance of two rings above and two rings below the limits of the foil. Tracheal gangrene developed not only in the segment isolated from its bed and wrapped in foil but also in the one or two additional rings above and below, which were surrounded by the sponge alone.

From this data our previously stated fourth axiom is proved, and it can be stipulated that a satisfactory result cannot be expected

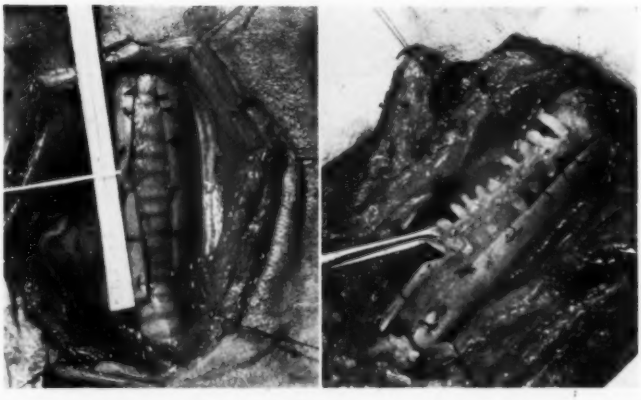

Fig. 5.—A. Decalcified section of bone replacing posterior tracheal window. B. Necrosis of anterior longitudinal segment of trachea same demonstrated in A.

from telescoping the trachea into a recipient tube, for if this is done the rings so introduced tend to slough in which case the implant will separate and come to lie free in the neck (Fig. 4).

These observations have completed Stage I of the experiment and provide much fundamental basic data but are in themselves simply a means to an end.

Stage II consists of observations upon the action and fate, not of artificial prosthetic-like materials, but of tissues of the living animal which might serve to replace the complete circumference of resected segments of the trachea without recourse to foreign substances. The first to suggest themselves as possibilities are the various hollow tubes of the body, such as segments of large blood vessels like the aorta or segments of the trachea itself from donor animals. These have been tried with but limited success.

My present report is based upon the use of bone, since it is obvious that a segment of long bone rasped free of marrow makes a non-collapsible tube with interesting possibilities. After one or two experiments it was decided to decalcify the bone, and specimens were prepared and used from flat bones such as the scapula as well as from long bones.

The first experiments with decalcified bone were successful and produced Axiom 5, which states that a resected portion of the anterior

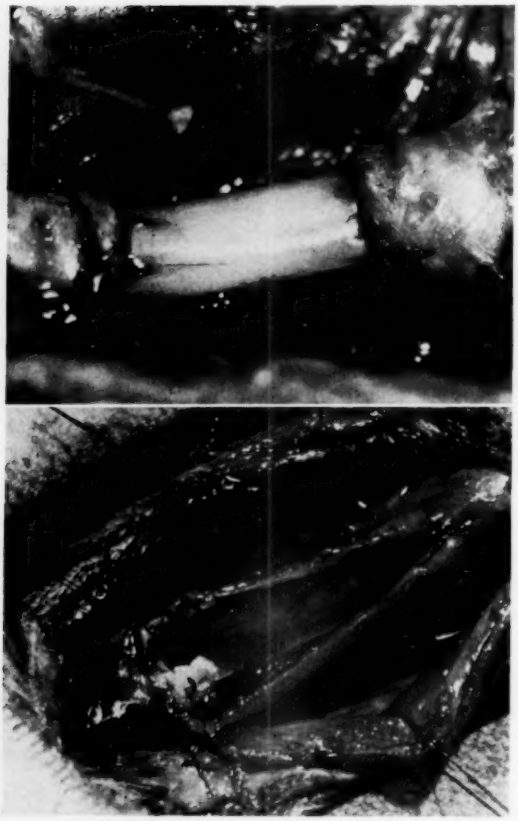

Fig. 6.—A. Decalcified bony tube replacing resected tracheal segment. B. Same trachea as "A" after resorption of bone and formation of fibrous tube.

or dorsal surface of the trachea even to the extent of half its circumference if covered with a section of decalcified bone as an insert, will heal satisfactorily with an adequate lumen. The remaining arc of trachea of the resected portion will survive because its blood supply has remained intact, the segmental vessels entering the trachea in the posterior quadrants.

Axiom 6: If, however, the resected window is posteriorly placed leaving only the anterior wall of trachea intact, failure will result

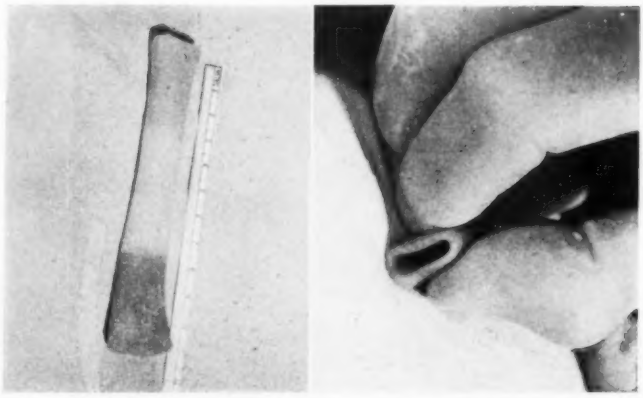

Fig. 7.—A. Rasped long bone with decalcified cuff. B. End view demonstrating elasticity of cuff.

since the remaining sector of trachea will necrose and slough due to loss of its blood supply which occurs by virtue of the details of vascular anatomy already described (Fig. 5).

Axiom 7: If a completely resected length of trachea is replaced by a segment of hollow bone, such as the femur which has been rasped out to form a tube and then decalcified, a fibrous tube will replace the decalcified bone; but this will not be sufficiently rigid to maintain a lumen. The decalcified bone itself is absorbed and replaced by fibrous tissue in the form of a non-rigid tube (Fig. 6).

These observations led to the final experiment to be completely reported upon today. Having established the fact that fibrosis takes place between a decalcified section of bone and the trachea, requiring only rigidity for a satisfactory result, it seemed to me that if just the ends of a hollowed out long bone could be decalcified as a cuff for purposes of establishing union and the remainder of the bone allowed to remain calcified, the desired end-result might be forthcoming (Fig. 7). Such bones were, therefore, prepared, and part of the decalcified cuff at each end was telescoped into the proximal and distal orifice of the trachea from which a complete segment of about 8 rings had been removed. The bones were then anchored with varying types of sutures.

In no instance did fibrosis between the tracheal ends and the decalcified bone take place, but quite to the contrary after five days

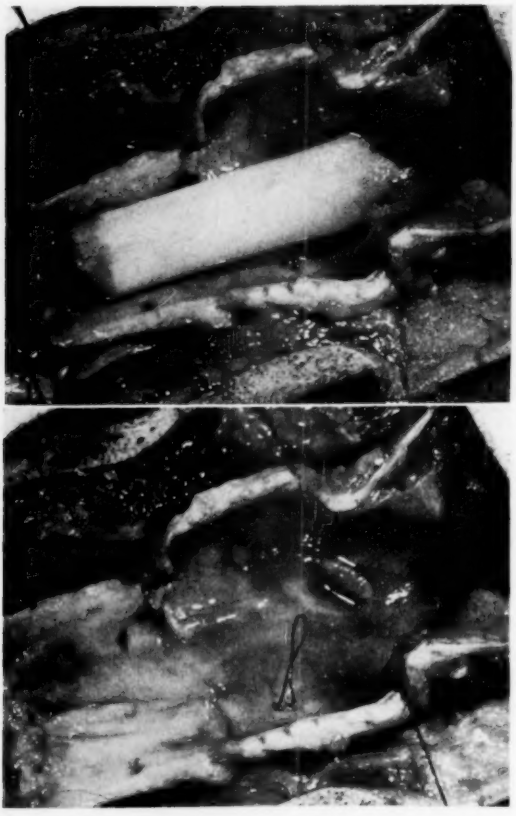

Fig. 8.—A. Free lying bony implant with decalcified ends, demonstrating absorption of decalcified segment after one week. B. Demonstrating fibrous tube formed about bony implant which has replaced resected section of trachea. The new fibrous tube appears in lower left hand area (no tracheal rings). Note that it is completely adherent to the trachea.

to a week the decalcified ends were completely absorbed, the calcified portion of bone remaining as a free insert within the thin fibrous tube that had formed about it (Fig. 8). Usually the free lying bony tube, being smaller in diameter than the trachea, was displaced either up or down, thereby permitting the fibrous pseudo-trachea to collapse with resultant respiratory occlusion.

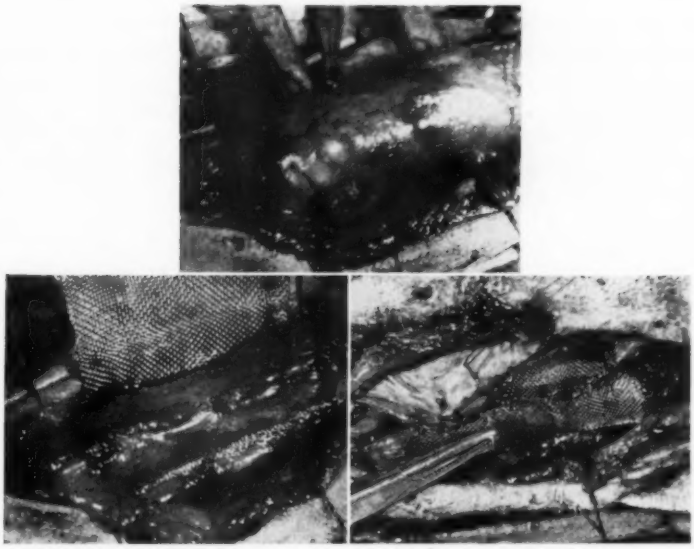

Fig. 9.—A. Flaccid tracheal segment which has formed about polyethylene tube. The latter removed by bronchoscopy. B. Polyethlene tube reinserted and fixed in situ with sutures. Beginning steel mesh wrap around. C. Wrap around completed. Deep fascia being sutured as envelope about steel mesh.

What has been accomplished by these experiments? First we have been able to repair large tracheal defects by the use of decalcified bone patches and other materials. Secondly it has been determined that in repair of any tracheal defect, the details of vascular anatomy are of prime importance. Thirdly we have learned to construct a fibrous tube lined with mucosa, but we have yet to learn how to provide it with adequate rigidity.

Two series of experiments have been begun and are awaiting observation and evaluation. The first is the use of a solid bony tube utilized in much the same fashion as the polyethylene tube. The disadvantage of such inserts even if successful is, of course, that a length of trachea is replaced by inert tubes without ciliated epithelial lining, the absence of which is of physiological importance.

In order to reconstruct a rigid pseudo-trachea lined with ciliated epithelium, advantage is being taken of previous experiments result-

ing in the formation of non-rigid epithelial lined tubes. Such previously formed tubes are being maintained patent by temporary polyethylene moulds within their lumens and various coarsely woven meshes, such as stainless steel, tantalum, and, possibly later, flexible nylons are being utilized as wrap-arounds. It now appears as though these previously flaccid tubes will adhere to the rigid or semi-rigid mesh and thereby remain patent. If this proves to be a reality, a rigid epithelial lined tube will have been successfully reconstructed (Fig. 9).

U.C.L.A. SCHOOL OF MEDICINE.

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DISCUSSIONS

DR. ALDEN H. MILLER (Los Angeles, Cal.): I must first of all say that I haven't had a bit of experience with experimental animal work in this field, but as I watched this work, I couldn't help but try to connect it in my own mind with some practical applications.

In association with Dr. Simon Jesberg, I have seen quite a few tracheas that had either been diseased following diphtheria, or had been injured following trauma, in which there was a lack of continuity of the lumen of the trachea.

I have handled two cases in which there probably was a separation of as much as a centimeter between tracheal rings, but where there was no loss of the tracheal rings themselves. We were able to establish a very good lumen with just the installation of either rubber or acrylic sections. We had a few sad experiences, though, in trying to maintain a lumen when there had been practically total loss of the cartilaginous rings, and I know of four cases in which we attempted to restore the rigidity that Dr. Pressman would like to get in the tracheal lumen, by just cartilaginous implants. Of those four, at least two were total failures, and the other two were very poor excuses for successful operations.

We have just had a patient at Children's Hospital in which there was total loss of any lumen in the trachea for a distance of almost 2 centimeters. We inherited this case, and all we could prove by dissection of that trachea was that there was a solid block of fibrous tissue with just two or three little kernels of

cartilage left. By dissecting out a channel and using both acrylic and rubber catheters (when we ran out of the acrylic one day, we used part of a rubber catheter) we did establish a gutter that had a good posterior wall.

Maybe it doesn't quite fit his work here, in which, in most cases, he completely removed the whole circumference of the trachea, but, having had some poor experience with cartilage implants before this time, I asked our plastic surgeons, "Can you build me a roof for this gutter?" There was no cartilage at all; the anterior wall collapsed with any attempt at making a complete closure.

They first inverted a flap of skin horizontally. Then they placed tantalum mesh over the flap and then brought up another flap of skin from the chest as the outer covering.

Just a week before I came here—which was about three months after the operation—I passed a bronchoscope, and so far we have a beautiful lumen, almost 50 per cent bigger than a 7 millimeter bronchoscope in this 8-year old child, so maybe we are going to have some success.

Of course, that was probably just a small separation of tracheal rings, and a small loss of the cartilaginous framework, and ,of course, it may be that now Dr. Pressman will very soon be able to show us that his decalcified bone will do even better than the plastic skin flaps. Certainly, this has been a wonderfully stimulating piece of experimental work.

Dr. Herman J. Moersch (Rochester, Minn.): Dr. Pressman's excellent paper has been most stimulating to me because of our interest at the Mayo Clinic in the problem of surgical resection of tumors of the trachea. The experimental work which my associates, Dr. Grindlay and Dr. Claggett, and I have carried out on resection of the trachea in general agrees with that described by Dr. Pressman. We found that one of the most important factors in sectioning the trachea was the tendency of the severed trachea to retract and cause tension at the site of anastomosis. In the experimental animal, it is possible to resect from 5 to 7 cartilaginous rings and restore the continuity of the trachea. In the human being, it usually is not possible to resect so large a segment of the trachea and re-establish its continuity. This is probably due to the fact that the dog has a much longer neck than the human being, and the head of the dog can be more acutely flexed than that of the human being, thereby relieving some of the tension on the trachea.

As Dr. Pressman pointed out, granulations do tend to occur about the end of tubes that are used to bridge defects in the trachea. In our opinion, this is more likely to occur if the tube does not fit the severed end of the archea snugly, or if the tube is not well anchored. We have observed well-fitting polythene tubes, used to replace a section of the trachea, that had been in place for a year or more with very little granulomatous formation about the end of the tube.

If possible, it is advisable to maintain a section of the tracheal wall when a tumor is resected, as this prevents the trachea from retracting. We have resected as much as half the diameter of the trachea and restored the tracheal wall by either a fascia or skin graft, both over an inserted polythene tube and without such a tube.

As Dr. Pressman has so clearly pointed out, the essential point in obtaining satisfactory healing in operations on the trachea is to remember the source of the blood supply, and that if the posterior half of the wall of the trachea is removed the blood supply to the anterior half will be eliminated.

Dr. Claggett has now operated on 4 patients with tumors of the trachea, 2 with carcinoma, and 2 with cylindroma. The 2 patients who had cylindroma have responded very well. One of the patients who had carcinoma lived for a year or more after operation and 1 died at the time of operation. It is a procedure that offers a great deal, especially if the diagnosis can be established early.

Dr. Pressman is to be congratulated on his splendid presentation, and I am sure that the information he has given us will go a long way in advancing our knowledge of the successful treatment of tracheal tumors.

DR. JOEL J. PRESSMAN: I think that perhaps before thanking the discussers, I should point out the practical applications of the experiments, especially those portions dealing with resections of less than the complete diameter or circumference of the trachea. Our speciality is expanding in the direction of more and more neck surgery, and carcinomas involving the thyroid gland for example are beginning to play more and more of a role in surgical fields that we are called upon to cover.

When the tracheal wall is involved the need to reestablish its continuity is obvious and these experiments are designed to help us meet such problems. Likewise in trauma from various sources the need for tracheal repair is obvious.

Dr. Miller's clinical experience with the case he recited brings out the practical application of experimental evidence that I have tried to indicate, namely that the posterior quadrant of the arch of the trachea will survive, because it carries the blood supply. The anterior arch will not survive. Dr. Miller's experience with the posterior arch is one in which I feel very certain he will, with the collaboration of his colleagues, achieve a successful clinical result.

Dr. Moersch's remarks concerning the development of granuloma are certainly well taken. These granulomata do not occur in every case. They occur primarily because of the movement, but the trachea is a mobile organ, and there is no way to avoid tracheal mobility; it moves with each respiration. The implant can remain fixed only so long, and then it becomes mobile.

We have had dogs live for a long period of time without the development of granulomata. As a matter of fact, my colleague, Dr. William Longmire, has a case in a human who has survived for four years with a polyethylene implant. This case was the result of an automobile accident, in which the cervical trachea was involved.

The removal of a segment of trachea and the retraction of its distal end into the thorax is, of course, observed in every one of these experiments, and accounts for the fact that end-to-end suturing is so often unsuccessful. An end-to-end suture is subject to the strain of the constant movement of the trachea as it retracts into the thorax with each breath. End-to-end sutures are difficult to maintain satisfactorily.

I think perhaps the discussants have covered all of the points that I may make, and time being of the essence, I shall not continue with this, although the subject is one of great fascination, and I could go on for very much longer.

LXXII

EPIPHRENIC ESOPHAGEAL DIVERTICULUM

F. Johnson Putney, M.D.

AND

Louis H. Clerf, M.D.

Philadelphia, Pa.

Supradiaphragmatic esophageal diverticulum of the pulsion type is infrequent in occurrence and indeterminate in cause. Located in the lower third of the esophagus, commonly within a few inches of the hiatus, it projects more often into the right thoracic cavity than the left. During the last 150 years since the initial description of the anatomy and clinical symptoms by Deguise[1] major advances in the diagnosis and treatment have developed, but the etiology must still be considered unknown. In contrast to the pulsion type of lower esophageal diverticulum the traction diverticulum, which rarely produces clinical symptoms, is due to contraction from fibrosis caused by an inflammatory process of the outer esophageal wall associated with disease of the lymph nodes at the tracheal bifurcation and hilar region that have become adherent to the esophagus.

In the cases reported by Harrington[3] and Lahey[6] the relative rarity of epiphrenic diverticula was demonstrated by the fact that this lesion constituted between 3 and 4% of the surgical procedures performed for all types of pharyngeal and esophageal diverticula. At Jefferson Hospital there have been a total of 244 surgically treated diverticula, and 7 cases of epiphrenic diverticula; the percentage corresponds closely with those previously mentioned.

The pulsion type of diverticulum whether originating in the pharynx or the lower esophagus is caused by pressure from within and is essentially a herniation of the mucous membrane through the muscle coats. Distinction between the types of diverticula can be made by microscopic study of the musculature in the wall of the sac. On histologic section of epiphrenic diverticula all layers of the esophagus can generally be seen, but the muscle layer is thin and sparse, with consequent weakness of this layer (Fig. 1). In the traction

From the Department of Laryngology and Bronchoesophagology, Jefferson Hospital, Philadelphia, Pa.

Fig. 1.—Histologic section of normal esophagus and neck of sac. Transition from muscle bundles in the normal esophageal wall to absent musculature in the sac wall is abrupt.

type the musculature is well-developed and normal in amount. In addition, histologic study of the surgical specimens of 3 cases of epiphrenic diverticula revealed decreased muscularis mucosa, with edema and hemorrhage in the stroma (Fig. 2). Disturbance in the sympathetic nerve ganglia has been advanced as a contributory cause, but in these sections the ganglia were found intact without histologic abnormality.

Several theories of why the muscle layer is weak have been advanced, beginning as early as 1833 when Mondiére[7] related 3 possible theories: (1) separation of the muscle fibers, probably congenital, (2) prolonged sojourn of a foreign body and (3) any obstacle whatever to passage of food. In spite of this and other conjectures when an epiphrenic diverticulum exists without an associated obstructing lesion no explanation of the origin can be substantiated.

Fig. 2.—Histologic section of wall of epiphrenic diverticulum. The muscle coat is sparse and practically non-existent.

Epiphrenic diverticulum is frequently associated with other abnormalities of the lower esophagus, chiefly achalasia and hiatal hernia. In 1952 Goodman and Parnes[2] found 128 cases sufficiently described in the literature to be reliable, and 65% were associated with achalasia. Also included in this group were 8 cases previously reported by Holinger,[4] 6 of which were associated with hiatal hernia. Kausel and Lindskog[5] found as much evidence to consider achalasia secondary to the diverticulum as in the reverse order. In those cases complicated by hiatal hernia the mechanism of the development of the diverticulum still is not clear.

Lower esophageal pulsion diverticulum presents no characteristic symptoms. Seldom are the symptoms clear-cut, and in many cases the diverticulum is an incidental finding in the patient with digestive disturbances. The symptoms progress with increase in the size of the sac, which may become large enough to produce pressure and

distortion of the subdiverticular esophagus with resulting obstructive symptoms. The symptoms are indistinguishable from those of other lesions affecting the lower end of the esophagus. When the diverticulum occurs in association with achalasia or hiatal hernia it is difficult to know which lesion is actually producing the symptoms. Dysphagia is the principal complaint, but epigastric distress, regurgitation, anorexia, nausea, loss of weight, regurgitation of blood, and cough also occur. Only occasionally does regurgitation of undigested and decayed food cause the distress that it commonly does in the pharyngo-esophageal diverticulum.

The roentgenographic picture of epiphrenic diverticulum may be confused with achalasia in which there is a large sigmoidal dilatation of the distal esophagus. Carcinoma of the lower esophagus may produce a false diverticulum above the carcinomatous stenosis, but generally there is a uniform saccular dilatation in which the entire circumference participates. An associated hiatal hernia may be difficult to diagnose roentgenologically, for the large amount of opaque material commonly used to demonstrate the diverticular sac often obscures the mucosal pattern in the subdiverticular esophagus which is necessary to detect the hernia.

Esophagoscopy serves chiefly to confirm the roentgen findings and discover complicating factors. The possibility of malignancy in the fundus of the sac must always be considered, and four cases of true epiphrenic diverticulum associated with carcinoma in the sac have been reported. Investigation of the subdiverticular esophagus is equally as important as inspection of the diverticulum itself. Considerable difficulty may be encountered in entering the stomach, but additional help is afforded by either utilizing a swallowed thread as a guide or by insufflation of air to iron out the lower esophageal folds. Because of distortion incident to the diverticulum, associated lower esophageal lesions escape detection in some instances. These diverticula have large mouths and the proximal lumen leads directly into the dilatation, so that the esophagoscope commonly enters the sac first. Careful inspection after withdrawing the instrument for some distance is needed to locate the true esophageal lumen. Accurate localization and determination of the size and extent of the sac provide additional information. When the site of origin of the diverticulum is located and it can be ascertained to which side the fundus projects the surgeon receives valuable aid in selecting the most accessible surgical approach for exposure. The presence of retained food and esophagitis should be eliminated before surgical treatment, if possible.

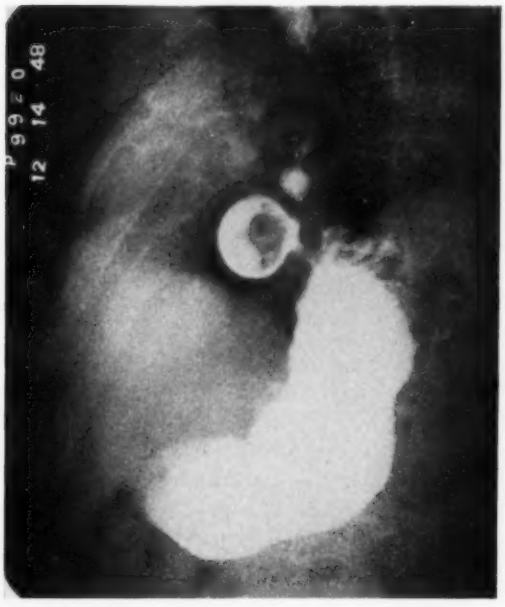

Fig. 3.—Recurrent diverticulum after surgical excision. Resection of lower esophagus with esophago-gastrostomy was performed at second operation. Post-operative esophagoscopy revealed fluid retention, mucosal redundancy and dilatation.

A small diverticulum can be treated conservatively with careful attention to diet, and, if asymptomatic, no treatment is required. When associated with achalasia dilatation of the hiatus affords only partial and temporary relief. As the diverticulum enlarges, retains food and produces secondary esophageal obstruction, surgical treatment is needed. The method of surgical treatment is variable, and in the four operated cases in this report diverse procedures were performed. In two simple excision of the sac was performed; in another with an obstructing lesion present esophago-gastrostomy with excision of the sac and lower end of the esophagus was done; and excision of the sac with repair of a hiatal hernia and Heller procedure for achalasia were performed in the fourth case when three coexisting lesions were found.

It has been our experience that when there has been any surgical interference with the esophago-gastric junction complications de-

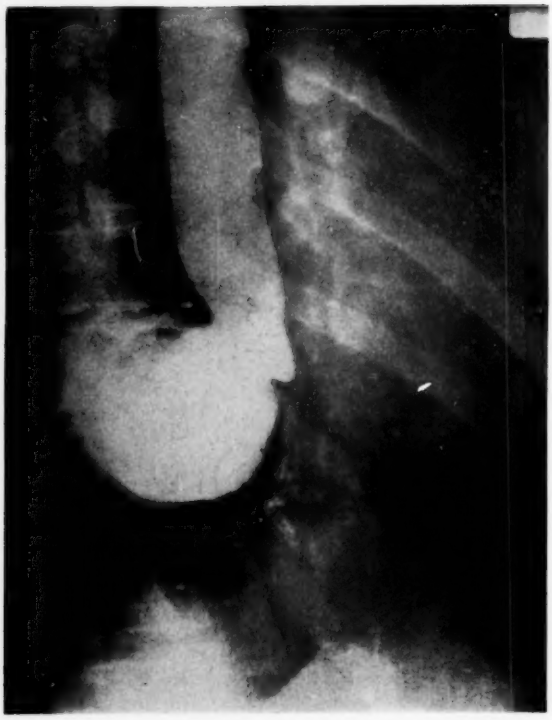

Fig. 4.—Large epiphrenic esophageal diverticulum in patient with associated hiatal hernia and achalasia. Treatment was surgical excision of the sac, repair of the hiatal hernia and Heller operation for achalasia.

veloped, the most alarming of which has been repeated and severe hemorrhage. On esophagoscopy esophagitis was almost invariably found. From these observations surgical procedures which disturb this relationship appear contraindicated in uncomplicated cases.

REPORT OF CASES

CASE 1.—A 66 year old refugee had attended many European clinics, and after the diagnosis of esophageal diverticulum had been made he was operated upon by Sauerbruch. The symptoms of tightness in the chest and lower esophagus on deglutition, and regurgitation at times persisted following the surgical treatment. Roentgen study revealed an epiphrenic diverticulum (Fig. 3). On esophagoscopy

Fig. 5.—Photograph of sac after excision.

the diverticulum was found to be large, communicating with the esophagus by a wide mouth. The sac projected to the right side, and exhibited chronic esophagitis. At operation Dr. J. H. Gibbon, Jr., found considerable scarring and narrowing above the diverticulum and resected the lower 5 cm of the esophagus with the diverticulum and performed an esophago-gastrostomy. Roentgen examination and esophagoscopy 17 days after operation revealed an unobstructed esophagus with no narrowing. When reexamined four years later there was found marked esophageal dilatation, retention and esophagitis.

CASE 2.—A 44 year old white housewife experienced difficulty in swallowing for 6 years. The beginning of the swallowing difficulty occurred with pregnancy. One year later the diagnosis of cardiospasm was made, and she received her first dilatation which was repeated at intervals. During the 6 years she would have attacks of esophageal

Fig. 6.—Large diverticulum with approximately 2 cm mouth. Subdiverticular esophagus is compressed and distorted. Surgical excision of sac was performed.

obstruction, and for 8 days prior to hospitalization was unable to eat anything. The roentgenologist could not be certain whether the dilatation was due to an epiphrenic diverticulum or a large hiatal hernia (Fig. 4). On esophagoscopy the entire esophagus was dilated, particularly in the lower third, where the folds were numerous, thick and leathery. Above the hiatal level a diverticulum was found, originating from the right lateral and posterior wall, opening by a large mouth into the esophagus. With the help of a previously swallowed thread the subdiverticular esophagus was inspected and appeared normal. However, at operation by Dr. K. E. Fry in addition to the diverticulum

Fig. 7.—Diverticulum associated with hiatal hernia in a 47 year old patient. Chief symptom was substernal pain upon taking food. Surgical treatment was declined, and 17 years later patient is still carrying on his usual activities without undue distress provided the diet is watched.

there was found a hiatal hernia and an achalasia. Excision of the diverticular sac (Fig. 5), reduction and repair of the hiatal hernia and the Heller operation for achalasia were performed.

CASE 3.—A 51 year old white autopainter had experienced vague digestive disturbances for sometime, and a roentgenogram revealed a diverticulum in the lower esophagus (Fig. 6). At esophagoscopy a large pouch communicating with the esophagus at a point 36 cm from the upper alveolar margin was found. The opening of the sac was at least 2 cm in diameter and was given off from the left lateral wall. The pouch contained a large quantity of undigested food in varying states of decomposition. The mucosa of the sac was thickened and the seat of a chronic esophagitis. At operation by Dr. T. A. Shallow the large diverticulum was dissected from the surrounding structures at the lower end of the esophagus and removed.

CASE 4.—For 4 years a 47 year old physician experienced sharp attacks of pain under the body of the sternum radiating to the left, which occurred on taking food. This was associated with a choking sensation. He also noticed that in the mornings he would regurgitate a mouthful of sweet, frothy material which never contained food particles. At the time of the onset of the symptoms a barium swal-

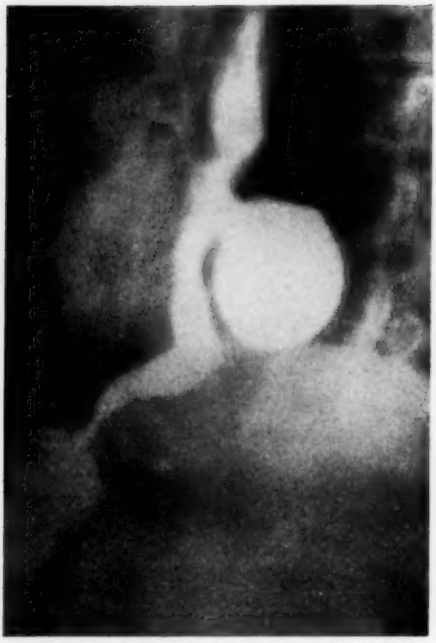

Fig. 8.—Dysphagia, noisy deglutition, regurgitation and substernal pain for many years were the chief symptoms produced by this diverticulum. Because of advanced bilateral tuberculosis surgical treatment was not performed. The patient learned to empty the sac by posturing herself and was able to continue her daily routine.

lowing function was reported as normal. Several months later he began to have pain on eating so that swallowing was impaired. There was a sensation of food going up and down, and the patient would attempt to wash it down with large quantities of fluids. Vomiting did not occur. The pain could be relieved by taking belladonna before meals.

A roentgenogram taken about 4 years after the onset of symptoms suggested 2 lower esophageal diverticula, one arising from the posterior wall just above the hiatus and the other from the anterior wall at the hiatal level (Fig. 7).

On esophagoscopy at a point 39 cm from the upper teeth there was found a pouch of the esophagus extending to the right. A broad

Fig. 9.—Diverticum producing pressure upon the subdiverticular esophagus and interfering with passage of food into the stomach. On esophagoscopy the sac contained retained food and moderate esophagitis was present.

spur with its axis from one to seven o'clock was noted between the pouch and the esophagus. The sac had a wide mouth, contained food, and the mucosa was inflamed and thickened though not ulcerated. The sac was approximately 5 cm in depth with an orifice at least 2 cm in diameter. An associated hiatal hernia was also observed.

The patient did not consent to surgical treatment, and a follow-up study performed 17 years later revealed little change in either the diverticulum or the hiatal hernia. He is able to continue his daily activities, but it is necessary for him to follow a semirestricted diet.

CASE 5.—A 67 year old white female with bilateral apical tuberculosis experienced dysphagia for both liquids and solids intermittently for several years. Noisy deglutition and regurgitation were

Fig. 10.—Large epiphrenic diverticulum in patient with increasing dysphagia for 6 months. Esophagoscopy revealed stenosis and tortuosity with obstruction of the subdiverticular esophagus. In addition to the small traction diverticulum, a small pharyngoesophageal diverticulum was also present.

also present. Substernal pain and a sensation of pressure occurred at times, but there would be intervals in which the patient was entirely symptom free. Roentgenographic study revealed a diverticulum of the esophagus a few inches above the diaphragm (Fig. 8). On esophagoscopy the opening of the sac was found to be about 2 inches in diameter with the fundus of the sac projecting posteriorly and to the right. The subdiverticular esophagus was patent, and no obstruction was encountered in entering the stomach.

Because of the advanced pulmonary tuberculosis surgical treatment was not performed. The patient learned to empty the diver-

ticulum by posturing herself and was able to obtain sufficient nourishment to resume her daily work.

CASE 6.—For 6 months a 52 year old white male complained of dysphagia for both liquids and solids. Regurgitation occurred at times, and soreness with lodgment of food in the mid-thoracic region was a prominent symptom. Noisy deglutition was also present.

The roentgenogram revealed a large esophageal diverticulum measuring approximately 2½ inches in its vertical diameter and 1½ to 2 inches in the lateral diameter. The diverticulum was located just above the diaphragm and projected into the right side of the thorax (Fig. 9). The sac was found to empty when the patient was on the left side, but the barium solution ran back up the esophagus rather than into the stomach. When the patient was rotated on his back the barium again entered the pouch but very little entered the stomach. Associated spasm of the esophageal hiatal was also noted.

On esophagascopy the diverticulum was found to be large, contained retained material, and moderate esophagitis was observed. There was no organic stenosis or ulceration. Both the subdiverticular esophagus and hiatus appeared normal.

The patient decided against surgical treatment and followed a rather strict dietary regimen for many years.

CASE 7.—A 51 year old white female experienced difficulty in swallowing pills for 6 months. Medication was being administered for rheumatoid arthritis, and the dysphagia was attributed to this condition. Three months later difficulty in swallowing solid foods developed, and after taking several swallows a feeling of fullness beneath the lower sternum was noticed. The fullness was relieved by eruption of gas. The symptoms progressed so that there was dysphagia for soft foods also, with a loss of 10 lbs. weight in the 3 month period preceding her hospital admission.

Roentgenologic study revealed a large epiphrenic esophageal diverticulum (Fig. 10). An associated small, lower esophageal traction diverticulum and a small pulsion pharyngoesophageal diverticulum were also found.

On esophagoscopy the sac was found to be the seat of a moderate degree of esophagitis, but there was no retention of food. The subdiverticular esophagus was narrow and stenotic, indicating an obstructive lesion.

The treatment was surgical excision of the sac.

SUMMARY

Seven cases of epiphrenic esophageal diverticulum are reported. Histologic study of the walls of the excised diverticula revealed the muscular coat to be thin and practically absent, but the sympathetic nerve ganglia were intact without microscopic abnormality. There were no characteristic symptoms of this lesion. One case had an associated hiatal hernia, another was complicated by both a hiatal hernia and achalasia, and a third had coexisting traction and pharyngoesophageal pulsion diverticula. Although the diagnosis was made by roentgenologic study esophagoscopy served to confirm the diagnosis and detect complicating lesions, at the same time providing additional information about the mucosal changes. Three patients were not operated upon, and all were able to perform their daily routine, one patient having a 17 year follow-up period. In the surgical removal excision of the sac without interference with the esophago-gastric junction is preferred over resection of the lower esophagus and esophago-gastrostomy because of the subsequent development of esophagitis and dilatation of the esophagus after the latter procedure.

1530 Locust St.

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DISCUSSIONS

I can agree with Dr. Putney in most of his concepts. I have also found achalasia associated with diverticula and have always thought that one cause of any diverticulum is some obstruction and stress in getting food through the esophagus. There would be no reason for the pulsion type of diverticula unless excess pressure was required to force food through. I would also agree that there are cases in which we do not have a definite visible cause.

Dr. Putney's method of insufflating air into the esophagus during examination should be stressed. I am amazed to find that quite a number of esophagologists fail to insufflate the esophagus during an examination. An inflated esophagus is dilated or abnormally large esophagus, much better than you can in the non-inflated esophagus.

Also, I would like to reinforce his statement about the interruption of the valvular action at the esophagogastric junction. It is a good thing not to remove this area. We see too many cases of esophagitis resulting from the destruction of the esophagogastric valvular action.

Dr. Louis H. Clerf (Philadelphia, Pa.): That old adage that "ignorance is bliss—it is folly to be wise," served me well in my early cases of epiphrenic diverticula. I did not know that this entity existed and considered them as traction diverticula.

I wish to comment on the use of inflation of the lower esophagus referred to by Dr. Hoover. In my experience, examination of that lower fourth of the esophagus is a difficult part of esophagoscopy. The use of the window plug and the hand bulb using gentle inflation has aided me more than anything I can think of in this particular portion of the food passageway.

I am heartily in accord with the statement made that the less one interferes with the sphincter action of the lower end of the esophagus, the less one's trials and tribulations will be.

LXXIII

GRASSES AND CEDAR TWIGS AS FOREIGN BODIES IN THE BRONCHI AND LUNGS OF CHILDREN

PAUL L. MAHONEY, M.D.

LITTLE ROCK, ARK.

Everyone knows that children have a habit of putting all kinds of foreign bodies into their mouths. Pieces of grass and twigs are no exception. For this reason, it would seem that this type of foreign body would be a very common one found in the tracheobronchial tree. However, a review of the literature gives the impression that just the opposite is true.

Recently Jackson[4] stated that the total number of cases of inspired grasses in the tracheobronchial tree is thirty-five. The pathological progress and the clinical features justify the conclusion that there are two distinct classes of cases. One class may be appropriately called the lodging type and the other the extrusive or one-way-travel type. Twenty-eight of this number were classified as the extrusive type and seven of the lodging type. In 1949, I published an article "Lung Pathology and Hemoptysis in Children, The Effect of Non-Opaque Foreign Bodies." If rice and wheat heads are to be classified as grasses, the two which I reported in this article would increase the total of the lodging type to nine cases. Probably many cases have not been reported in the literature because of either neglect or indifference.

Clerf[1] says that he has not seen a case of spontaneous perforation of the thorax by a grass head and that in a number of cases involving timothy grass heads there seemed to be no tendency to perforation. One of my clinical case reports is that of a boy who had choked on a twig of cedar. Apparently in time it would have made its way to the outside.

In none of the five clinical cases here reported was the foreign body visualized or removed bronchoscopically. Jackson reports that in the seven cases of the lodging type reported the foreign bodies were all removed bronchoscopically.

REPORT OF CASES

CASE 1.—White, male, 10 years old, admitted to St. Vincent's Infirmary July 24, 1939. His chief complaint was chronic, persistent cough with recent expectoration of large amounts of blood. For some months expectoration was streaked with blood, but recently the bleeding became rather profuse. The boy was in very good health until eighteen months before, when, while playing in a small cedar tree, he suddenly became choked on a small piece of cedar he had in his mouth. Gagging and choking excited him so that he fell from the tree, injuring his side. He was taken to a physician who ignored the history and proceeded to strap his chest, stating that several ribs had been broken by the fall. He assured the mother the twig had either been swallowed or coughed out.

Soon afterward the child developed a cough and produced sputum which at times was streaked with blood. The physician prescribed for the cough. Not until eighteen months following the accident, when the bleeding became profuse, was the child brought to Little Rock and admitted to a hospital. The boy was not critically ill, and a general physical examination was negative. A diagnostic bronchoscopy was done and the entire tracheobronchial tree was visualized as far as possible through a bronchoscope. Scant, foul-smelling, blood-tinged, secretions were aspirated for study. A small amount of iodized oil was instilled for the purpose of bronchography.

Two films of this patient's chest were taken on July 24, 1939, one a posterioanterior, the other a left lateral exposure. These films showed iodized oil injected into one of the branches of the left main bronchus reaching the anterior inferior part of the left lower lobe. Located very near the surface of the lung there was a spherical area of iodized oil about 1.5 cm in diameter. The film suggested that there might be a small excavation at that location. Except for this one small area, these films were entirely negative for signs of infection or other disease. The cause for this finding was unknown.

Surgical treatment was recommended but refused, and the child returned home. A few months later, the child expelled a piece of cedar following a violent coughing bout, and subsequently made an uneventful recovery. The piece of cedar was sent to me, and it appeared as though it had just been removed from the tree. I have seen this boy several times the past few years and he has had no further respiratory symptoms.

Had bronchoscopy been done early it is highly possible that the foreign body would have been visualized and removed. The mother

told the physician what was wrong, but acting on impulse rather than good judgment he ignored the history and instituted symptomatic treatment. The child paid the price.

CASE 2.—White, male, 5 years old admitted to the Arkansas Children's Home and Hospital in November, 1944. Chief complaint was cough of several months' duration. The onset was not unusual according to the physician who was called to treat the child and who referred him to the hospital for further study. All routine examinations were done and a diagnosis of chronic tracheobronchitis was made. Treatment was prescribed and the child was discharged from the hospital one week later.

In May, 1946, the child was re-admitted with fever, cough, and pains in the right side of the chest. Dr. John Agar did a diagnostic bronchoscopy and reported the right main bronchus to be filled with foul-smelling, thick pus. The mucous membrane was thickened, inflamed, and a small amount of blood was noted. A diagnosis of bronchiectasis was made, treatment was instituted, care outlined, and the child was discharged from the hospital with instructions to return for further observation.

The third admittance was December, 1946. The cough was very productive; the sputum was blood-streaked, and had a very foul odor. I was called in consultation January 1, 1947. After a review of the chart I repeated the diagnostic bronchoscopic procedure. Following aspiration, iodized oil was instilled for bronchography. After a review of these films a lobectomy was recommended. This procedure was carried out by Dr. Harvey Shipp on January 16, 1947, and a rice head was recovered at this time.

When the rice head was recovered at the time of operation and later shown to the parents and an older brother, the true story concerning the etiology was confessed. Several months preceding the child's first admittance to the hospital, he and his brother were riding on a rice wagon against the advice of their father, and it so happened that the younger child aspirated a rice head and nearly choked to death. Having been warned that they would receive a good whipping if they were ever caught riding rice wagons, they had never divulged the incident to their parents. In this particular case it was impossible to obtain an accurate history at a time early enough to make proper recommendations.

CASE 3.—White, female, 10 years old, admitted to St. Vincent's Infirmary August 8, 1947, with the complaint of expectoration of large quantities of blood. She gave a history of being in good

health until three months before when she began having an elevation of temperature, as high as 104° F., accompanied by nausea. She had an appendectomy at this time with no relief of symptoms. About two weeks later she expectorated a small amount of blood, and then developed a nonproductive cough which persisted until the date of admission to St. Vincent's Infirmary. At this time she was expectorating large quantities of blood. The mother reported that her daughter had been returning home from school accompanied by her teacher, and that she had a wild wheat head in her mouth which she either aspirated or swallowed. She imediately began gagging and choking but shortly thereafter her symptoms abated and her mother was advised by a physician that the child had simply choked on the piece of wheat and swallowed it. This was about one week prior to onset of nausea and temperature elevation.

On the date of admission to the hospital the child was pale, cyanotic, and expectorating considerable blood. There was dullness to percussion over the right lung base, and rales were heard in this area. The hemoglobin was 49.8 per cent; R.B.C. 2,800,000; W.B.C. 17,300; temperature 104° F; pulse rate 160. She received blood transfusions, plasma, and penicillin.

Four days after admission a diagnostic bronchoscopy was done and blood was seen to becoming from the lower right lobe. Lipiodol was instilled. X-ray showed an abscess cavity in the base of the right lung along the mediastinal aspect and below the lung root. After several days of treatment the child improved sufficiently to permit operation. Dr. Henry Hollenberg and his associate, Dr. Robert Jones, removed the right sixth rib and opened the chest. The lower lobe of the lung was found to be very adherent in the sulcus and posteriorly. In freeing this at the sulcus a small cavity was opened, and a wild wheat head, about one-and-half inches in length, immediately showed itself and was removed, apparently intact. A lobectomy was thought to be indicated prior to opening into the abscess, but inasmuch as only a small portion of the lower lobe appeared to be involved, a partial lobectomy was done.

Following operation there was a hematuria for three days which probably was the result of the blood transfusions. Otherwise she improved rapidly. On the fourth postoperative day a routine chest film was made. This showed the iodized oil still present in the cavity along the mediastinal aspect of the right lung with no noticeable change from the earlier film except for the operative defect.

One month later the child again was operated upon, and a portion of the ninth rib posteriorly was removed. The lung was ad-

herent to the pleura. The lung was opened bluntly in this area, and two dark flecks of oiled necrotic tissue were removed. A vaseline gauze wick with a metal marker attached at the end was inserted in the cavity for drainage. An x-ray film showed the marker to be in the center of the abscess.

After the operation there was moderate purulent drainage which gradually subsided. The drainage opening was allowed to fill in from within outward. The temperature and pulse remained within normal limits for a period of two weeks, and her general well-being returned to normal.

The child and the teacher knew that the cause of the trouble was the sucking of a piece of wild wheat head into the windpipe. They conveyed this information to the mother who, in turn, told the physician. Had the physician only paid more heed to this positive history, the child probably would have been spared the procedures enumerated.

CASE 4.—White, male, 12 years old. He was admitted to Baptist State Hospital on September 12, 1947, with a chief complaint of pain in the left anterior chest associated with swelling and partial paralysis of the left arm, accompanied by a moderately productive cough.

Ten months before admission to the hospital this patient contracted a severe upper respiratory infection which was diagnosed as influenza. This infection subsided, and the boy was able to go back to school approximately three weeks after the onset. His cough, persisted. Three weeks later he again had an attack of so-called influenza which persisted for two weeks. He returned to school and was able to stay in school for four weeks. An acute exacerbation of influenza again required his confinement to bed for approximately one month. Two weeks before admission he complained of pain in the region of the left breast and his parents noted a large, firm, tender mass in this area. His local doctor put him into bed for a short time. The mass increased in size and tenderness in ten days and caused a partial paralysis of the left arm.

On physical examination his temperature was 99.8° F., pulse 100, and respiration 26. The left chest showed evidence of a reddened, inflammatory mass beneath the medial portion of the pectoralis major muscle. Auscultation over this area, posteriorly, revealed the presence of moist crepitant rales. There was percussion dullness over this area in about the second to third intercostal space. There was

some edema of the pectoralis major muscle causing voluntary limitation of motion of the left arm. This was not a true nerve paralysis.

Laboratory studies revealed a white count of 18,900; differential of 3 stabs, 70 polys, 27 lymphs. The red count was 4,350,000 with a 75 per cent hemoglobin. Urinalysis was negative. X-ray examination revealed a widening of the hilar shadow on the left side with strands of fibrosis and hyperemia extending outward from this area. A bronchoscopic examination was done and purulent material was found draining from the left upper lobe bronchus. Lipiodol instillation was carried out and an x-ray examination was made. This revealed a massive cylindrical bronchiectasis limited to the lingula of the left upper lobe with abscess cavities lying adjacent to the anterior chest wall in the region of the inflammatory mass. Sputum examination revealed large numbers of staphylococci.

Incision of the tumor mass was made down to the intercostal muscle and biopsy of the tissue was taken and reported as edematous and inflammatory tissue. No frank pus was obtained. This boy was placed on aerosol streptomycin inhalation. His general condition improved and temperature returned to normal within approximately one week. The character of the sputum cleared and his cough disappeared. His appetite improved and he began to gain weight. Due to the fact that there was slight drainage and delayed healing of the incised tumor mass on the anterior chest wall, he was sent home for a period of two weeks, after which he was supposed to return for lobectomy.

This patient was re-admitted to the hospital on October 4, 1947, in an improved condition. His cough had subsided and he had no fever, but the bronchiectatic cavity still persisted. On October 7 segmental resection of the lingula was carried out without difficulty by Dr. Harvey Shipp. Lying within the bronchiectatic abscess against the anterior wall was found a cedar leaf approximately one inch in length.

Postoperative recovery was uneventful and without complication. The patient was discharged from the hospital on October 25, 1947. He returned to school on November 17. At the present time the patient is entirely well.

This was a case of a non-opaque foreign body lodged in the lingular lobe of the left lung, a most unusual site for a foreign body of this nature. History of the aspiration of this foreign body was not obtained until a week following admission to the hospital when the mother happened to recall the incident. This case again illus-

trates very well the need for careful history in all cases of persistent lung infection and for utilizing all available diagnostic procedures.

CASE 5.—White, female, aged 19. Admitted to St. Vincent's Infirmary the night of May 11, 1943, at the age of 10 years. The patient was expectorating large amounts of blood following a severe coughing spell. The general appearance of this child was good; however, she was very apprehensive.

The following history was obtained from the mother. When this child was about six years of age she expectorated a small amount of blood following a cold accompanied by cough. An examination at this time, including a sputum study and an x-ray of the chest, was negative. A diagnostic bronchoscopy was done and the entire tracheobronchial tree as far as could be visualized was entirely normal in appearance. At various intervals, without any apparent reason other than maybe a cold, she would cough small amounts of blood. Many general physical examinations, sputum examinations, and x-rays of chest were always negative. The child continued to develop normally, both physically and mentally.

A general physical examination at the time of this admittance was negative other than for moist rales over the entire right chest. A posteroanterior film of the chest showed accentuation of the pulmonary markings with a little haziness along them in the lower part of the right lung.

A diagnostic bronchoscopy was repeated, and the entire tracheobronchial tree, as far as could be visualized, was normal as it had been before. Scant secretions were aspirated for laboratory study. No tumor cells, acid fast bacilli, or fungi were present. There were no reasons to suspect the presence of a foreign body other than the hemoptysis of unknown origin. There was no wheeze, productive cough, history, or bronchoscopic or x-ray evidence. Because of fear of hemorrhage and death and the fear of being publicly humiliated by a hemorrhage, this beautiful young lady lived a very cloistered and protected existence. She refrained from any form of physical activity for fear of cough and bleeding; she remained away from any individual or individuals who showed evidence of a cold for fear of contracting it. She refused to accompany girls and boys of her age to swimming, dancing, or other parties. Nor would she attend athletic events for fear of bleeding and suffering humiliation. She attended private schools. The fact that she appeared normal both physically and mentally but continued to live in a small world of her own, secluded with her parents, directed criticism toward her

parents for not encouraging her to partake in all youth activities. All attempts to convince her that she would not bleed to death, but some day would discontinue these bouts, failed to make any change in her mode of conduct. Upon finishing the high school curriculum in a private school with no expectoration of blood in many months, she was sent to college in September, 1952. At the end of about five or six days she developed a cold associated with a severe cough which in turn was complicated with the expectoration of a very large amount of blood. During the paroxysm of cough she expectorated a twig of cedar about one inch in length, which had the appearance of having just been removed from the tree. Following this episode she then told me that when she was about four years old she was playing with a piece of cedar twig in her mouth when she became excited from some cause and began to choke, gag, cough, and could hardly get her breath. She remembers vomiting up a piece of cedar twig. Because she had no more symptoms, the incident was forgotten. It was about two years later that she expectorated a small amount of blood, but in all the histories that were taken over a period of these years no history of the aspiration of a foreign body was ever elicited.

She recently told me that it probably would have been coughed out long before this time had she permitted herself to cough. She had learned that lying flat would provoke a cough reflex; for this reason she always slept with her head elevated in an effort to prevent cough and bleeding.

Unless this large piece of cedar worked downward by the ratchet-like action of the respiratory and tussive elongation and diametric luminal enlargement, followed by shortening and diametric diminution of the bronchial lumen, it is difficult to understand how it could be present in a bronchus and not be visualized. (The ratchet-like, one-way travel of foreign bodies in the bronchi was first noted in relation to pins by Jackson, 1915). It is also difficult to understand how this foreign body could remain for so long and only occasionally produce sufficient pathological changes to cause bleeding in various amounts, how these pathological changes could be present and manifest symptoms without positive physical and x-ray evidence, and how it could remain so long without a series of pathological changes becoming manifest and making certain symptoms constant.

SUMMARY

1. Five clinical case reports are presented.

2. Two of the foreign bodies were expelled by the cough reflex and three removed surgically.

3. Four were from the right lower and one from the left upper lobe.

4. None were visualized or removed bronchoscopically.

5. Ages were 5 to 19.

6. Length of time of foreign body remaining in situ 3 months to 14 years.

7. There were no deaths.

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DISCUSSIONS

DR. CHEVALIER L. JACKSON (Philadelphia, Pa.): I am interested in commenting on this paper, both because of the importance of the subject and also because of the fact that the recent paper of Dr. Jackson, Senior, in The Laryngoscope, brought this question to our attention recently. I also recommend that article to you, in case you have missed it; it is a most interesting piece of work. It is Dr. Jackson, Senior's most recent contribution to the literature.

I think these foreign bodies are important because of their elusiveness and I think the practical point that is most important is to see to it that we do a skillful bronchoscopy at the earliest possible moment when there is a history suggestive of this kind of foreign body, because of this tendency that many of them show to work their way quickly down into the smaller bronchi. If you do get in with a bronchoscope soon after the accident, and with a bronchoscope small enough to go down into at least the lobar bronchi, you have a chance of getting hold of the foreign body and getting it out before it becomes lost in the periphery. Once it has gone beyond, we don't have the possibility of using fluoroscopic guidance such as we do with pins and other foreign bodies that show this ratchet action, because the foreign body is not opaque enough to be seen, so we either have to wait and hope it will be coughed up, or go ahead with a lobectomy. Certainly, in some cases there should be no delay in doing a lobectomy.

Groping and probing down into the segmental bronchi, hoping you will get in the right one to get hold of the foreign body, can be tried, of course, but it has been my experience that it is rather discouraging in these cases.

This is something to be kept in mind as a cause of hemoptysis. In the obscure cases of hemoptysis, foreign bodies of this type may easily be the explanation.

I think that Dr. Mahoney has done us a service in bringing these very good, practical examples of this problem to our attention. I think it is scarcely necessary for this group to be told that we must prick up our ears whenever we hear a history—or when we can elicit a history—of possible foreign bodies. If there is any vague suggestion that the patient has been chewing grass or cedar twigs, let's recall this very fine exposition of the problem that Dr. Mahoney has given us.

DR. LOUIS H. CLERF (Philadelphia, Pa.): It was my good fortune to remove a cedar twig, bronchoscopically, in the case of a child about two years of age.

The history is so important. There was a Christmas tree, a cedar tree. The child had a twig of the Christmas tree in its hand and was chewing on it. There was a severe choking attack, and that was sufficient evidence to warrant a diagnosis of foreign body, particularly when the patient develops atelectasis of the upper lobe of the right lung, or any lobe, for that matter.

I was able to see a tiny bit of the twig projecting from the orifice of the right upper lobe bronchus, and was able to remove it. I was amazed that it had remained in that location and did not penetrate deeper. It may have been due to the conformation of the foreign body or in the arrangement of the bronchial subdivision in the segmental bronchi.

I reviewed two cases many years ago in the literature. One of them concerned a cedar twig; the other was some other form of evergreen. Both moved on very rapidly and entered the pleural space. The surgeons removed both by external incision. One of them got there within 48 hours, which I thought was a reasonably good record.

I have also removed a few timothy heads. As has been stated, too often one is not aware that there is a timothy head present. One of the greatest surprises of my life concerned a child four years of age whom we were treating for bronchiectasis during the time when bronchiectasis was treated by bornchologists. This was an extensive right lower lobe bronchiectasis. I used an open-ended aspirator. That was fortunate because the open-ended aspirator, introduced into a small bronchus, engaged a timothy head, and it was removed. It did not clear up the bronchiectasis, but it certainly explained the etiological factor.

One has to be continuously aware of the possibility of foreign body in obscure pulmonary suppuration. Too often we close our minds completely to etiology when once we have made a diagnosis of bronchiectasis.

I was very grateful to Dr. Mahoney for bringing this to our attention, because we are meeting up with fewer foreign bodies, and it will not be very long until the younger men will have forgotten all about foreign bodies.

DR. PAUL G. BUNKER (Aberdeen, South Dakota): I enjoyed this paper very much, because of two similar experiences of my own, one of which verifies the rapid ratchet action Dr. Mahoney describes.

The first case was a small child, approximately two years of age. We found this small evergreen twig already well down into one of the basal segmental bronchi on the left side. It was only during deep inspiration that the end of the twig could be visualized. Fortunately, we were able to secure it and to bring it out intact, with the infant-sized forward grasping forceps.

The other case was an extreme emergency because of the laryngeal obstruction and spasm due to aspiration of the head of a tickle grass blade. This has a very long beard, and it was lodged in the larynx of this baby. I do believe I ever saw a foreign body case any nearer death than in this instance. It had only been present for a matter of aproximately a half hour, and I am sure that in another half hour the patient would have been dead had the foreign body not been removed.

DR. PAUL L. MAHONEY (Little Rock, Ark.): I think what Dr. Jackson said is really very timely. When you get this history, you had better not procrastinate and say, "Well, maybe it will cough it out," or "Maybe there is nothing in there," I believe I could have seen these foreign bodies had I been given the opportunity to look early enough; as time went by, they worked their way down to where I couldn't see them.

I might have been able to have seen the one in the upper lobe on the left side if I had used one of the telescopic types of new instruments to look in that region.

LXXIV

RADIOLOGIC AND BRONCHOSCOPIC OBSERVATIONS ON DIFFUSE NARROWING OF THE LUMEN OF THE TRACHEOBRONCHIAL TREE

ROY R. GREENING, M.D.

AND

JOSEPH P. ATKINS, M.D.

PHILADELPHIA, PA.

Since bronchograms usually are performed in patients with clinical manifestations of pulmonary disease, one expects to find bronchial abnormalities in a high percentage of such studies. Several years ago we became concerned over the number of apparently normal bronchographic studies being diagnosed in such patients. In an exhaustive review of these bronchograms, we were impressed with the large number of cases showing a consistent abrupt narrowing of the bronchi shortly after their origin from the main trunks. We have chosen to call this narrowing bronchial attenuation.

These patients fell into three main clinical groups.

1. *Patients with diffuse pulmonary emphysema.*

2. *Patients with asthma or asthmatic bronchitis and hypersensitivity states.*

3. *A less easily defined group presenting clinical features strongly suggestive of bronchiectasis.*

The clinical features of emphysema and asthma are well known and do not require further definition here. The third,, less well defined group present the following clinical features: They are usually young adults with long standing pulmonary symptoms and productive cough with sputum possessing no identifying characteristics. In addition, they usually have repeated bouts of pneumonia-like episodes, fatigue and a tendency to lose weight. The physical examination almost invariably reveals a wheezing type of respiration with a variety of rales. The auscultatory findings have been confused with

From the Departments of Radiology and Bronchoesophagology, Hospital of the University of Pennsylvania.

those heard in patients with bronchiectasis. These clinical findings for the most part, have been non-specific, so that initial diagnostic impressions included either bronchogenic carcinoma, tuberculosis, bronchiectasis, chronic lung abscess, asthma or chronic bronchitis.

Bronchoscopically, these three groups can be differentiated as follows:

1. The emphysematous patient shows diminution in the expulsive force of the cough, marked respiratory intrusion of the posterior bronchial walls and thickening of the bronchial mucosa. Although secretion tends to be scanty, when present, it tends to be whitish, mucoid and not too viscid.

2. The asthmatic patient presents a more forceful expulsive power with cough, extremely viscid secretion and expiratory intrusion of the posterior bronchial wall. The mucosa is usually pale.

3. The third group of patients with which we are particularly concerned, show little disturbance in the expulsive force of the cough, consistent mucosal hyperemia, less secretion than one would expect to find from the physical findings and no expiratory intrusion of the posterior bronchial wall.

There are no characteristic bacteriologic findings in any of these conditions.

The roentgenographic findings in asthma and emphysema have been well described elsewhere by Barden[1] and the bronchographic findings in these two conditions have been beautifully illustrated by DiRenzo.[3] It is therefore our purpose only to outline the findings in the third group of patients.

Conventional roentgenograms of the chest in these patients have not shown any distinctive characteristic findings and usually are thought to be normal. Thus far the only objective findings have been demonstrated by bronchography. We hope, however, to be able to determine physiological alteration in lobes or segments of the lung by some of the newer pulmonary function studies now under investigation by Comroe.[2]

Bronchograms are performed by placing a catheter in the trachea and thoroughly anesthetizing the bronchi to each lung by the use of topical cocaine, after proper sedation. Injection of the opaque is carried out under fluoroscopic control being careful to use small amounts of the media to avoid flooding of the bronchial tree. Spot roentgenograms are made of segments of the lung as the study prog-

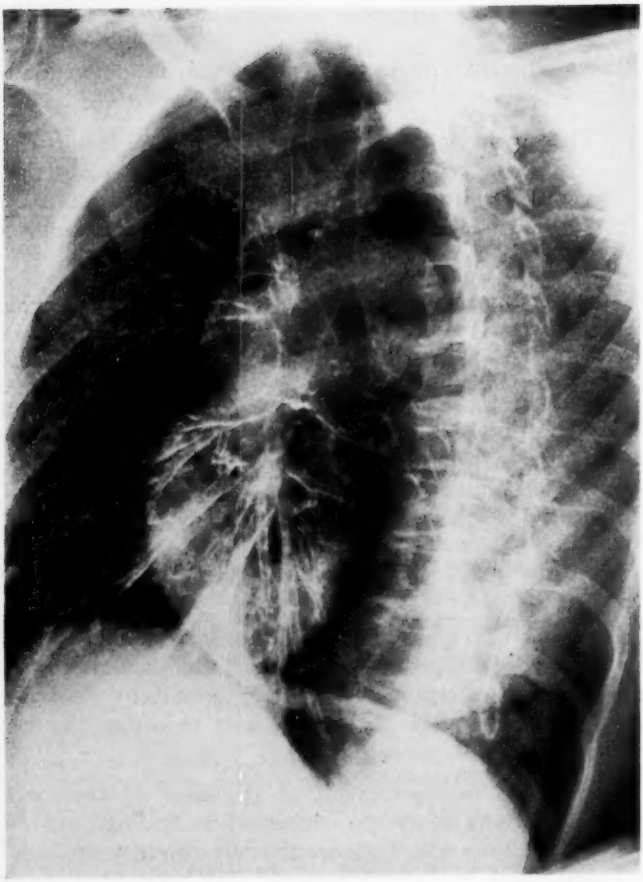


Fig. 1.—Normal bronchogram showing gradual decrease in size of the bronchial lumen down to the smallest bronchi. The spacing between bronchi is normal with all of the bronchopulmonary segments outlined. A small amount of alveolar oil is present.

resses and in certain instances supplemented by the use of full sized chest roentgenograms using the Potter-Bucky diaphragm. Essentially similar findings have been demonstrated by using iodized oil (iodochloral) or water soluble media (umbradil) as contrast agents.

The patients with bronchial attenuation show anatomic narrowing of the bronchi shortly after their emergence from the main bronchial trunks and wide spacing of these bronchi in the pulmonary pattern. Most important, however, is a physiologic alteration in the tracheobronchial tree manifested by variations in bronchial lumenal size in different pulmonary segments altering markedly the bronchopulmonary pattern. Such changes vary from segment to segment and from time to time in the same segment of the lung. During bronchography, difficulty and delay in bronchial filling will be found with prolonged retention of the contrast substance in the smaller bronchi of the affected segments of the lung. This results in almost no alveolar filling in these segments even with delays of twenty to thirty minutes after installation of the opaque media. Whether or not this physiologic alteration in the bronchus is an indication of an anatomical abnormality or a purely physiologic aberration is not apparent. Bronchodilator drugs thus far have not altered such patterns.

These findings are so striking, we now are looking for them in patients clinically suspected of having bronchiectasis whose bronchoscopic findings are not suggestive of this disease.

REPORT OF CASES

CASE 1.—B. S. This thirty-four year old white male was first seen in December of 1952. He had a twelve week history beginning with an upper respiratory infection, productive cough and chest pain. At the time of this admission he was complaining of cough and chest pain. During the first two weeks of this illness he had had hemoptysis. There had been an eleven pound weight loss, malaise, chills and a fever which was unrelieved by penicillin.

The initial roentgen examination of the chest was thought to be negative.

Bronchoscopic examination revealed concentric narrowing of the right upper, left upper and left lower lobe bronchi. No abnormal secretions were seen. The bronchogram revealed no evidence of bronchiectasis. Marked abrupt attenuation of the bronchi was present in almost the entire lung and there was wide separation of the bronchi with a marked delay in their filling and in the alveolar

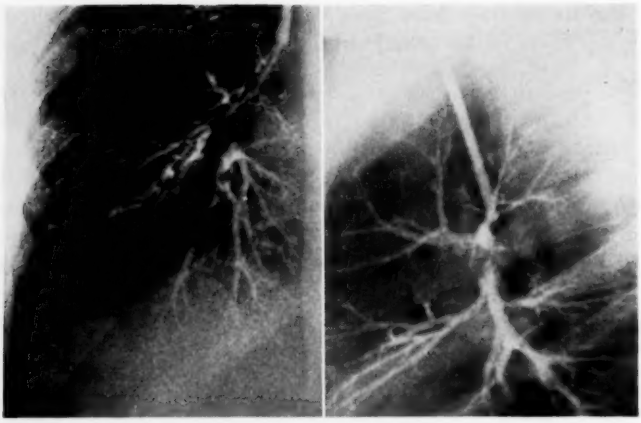


Fig. 2.—Bronchial attenuation: Spot roentgenograms of pulmonary segments made during bronchial mapping showing normal stem bronchi abruptly narrowing to widely separated filamentous bronchi with no alveolar filling.

filling. With the administration of a bronchodilator drug there was no alteration in the lumenal size of the bronchi at the end of a twenty minute period. This patient did not develop any dyspnea.

CASE 2.—H. A. The patient was a two year old white female who was first seen in 1949 after two months of wheezing thought to be due to a foreign body. In addition, there was a history of wheezing prior to the present illness and suggestive evidence of food allergies. On admission, the physical examination revealed a husky child with inspiratory and expiratory wheezes bilaterally, pale nasal mucosa and marked lethargy.

The roentgen examination of the chest revealed patchy areas of consolidation in the central portions of each lung raising the question of bronchopneumonia.

Bronchoscopy revealed intense hyperemia of the mucosa on the left side with no foreign body. The roentgen and bronchoscopic findings cleared with repeated bronchoscopic aspirations.

The patient was re-admitted two years later with cough, wheezing and several previous minor bouts of abdominal pain.

The physical examination revealed wheezing on both sides of the chest. Bronchoscopy again revealed signs of bronchial infection. The patient improved with penicillin, but developed an urticarial rash. There was a strong impression that we were dealing with an allergic diathesis, but all skin tests to the common allergens were negative.

The bronchogram revealed no evidence of any anatomical lesion except the abrupt bronchial attenuation seen in other patients with similar symptoms.

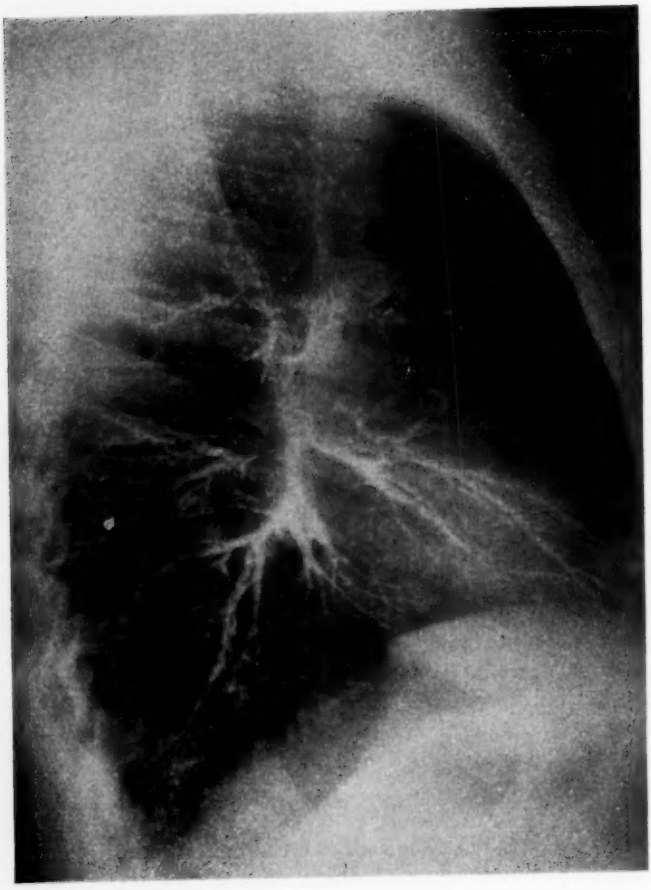
In spite of these findings we were still uncertain whether this child's symptoms represented a combination of an allergic state producing the bronchial attenuation or whether the symptoms were produced as a result of the bronchial attenuation.

CASE 3.—E. F. This 24 year old white male, was admitted in July 1951 with a previous history of virus pneumonia six years ago. He did not think that he became completely well and was discharged from the Army. The chest roentgenograms at that time are reported to show slight cloudiness. At the time of his admission to this hospital, he had morning cough, productive of one half cup of thick, foul sputum; a sensation of pressure on the left side of his chest, fatigue, shortness of breath, five pound weight loss, fever and night sweats two to three times monthly.

Physical examination: Pulmonary wheezes throughout chest and moist rales bilaterally in the bases. The roentgen examination of the chest showed prominent truncal markings at both bases possibly due to bronchiectasis. Bronchoscopy showed mucosal hyperemia with scanty, mucoid secretion. The bronchogram revealed no evidence of bronchiectasis. Many of the smaller bronchi show abrupt narrowing and attenuation in each portion of the lung. This changed somewhat from time to time.

CASE 4.—F. O'D. The patient was a 51 year old white male with a productive cough for years which became much worse five years prior to admission. At the time of admission, he had weakness, lethargy, paroxysmal non-productive cough, loss of twelve pounds in weight, and a right maxillary sinusitis of two years' duration.

The physical examination revealed a cloudy right maxillary sinus. The right side of the chest was smaller than the left. Otherwise the examination of the chest revealed a prominent left hilum with prominent markings on both sides.



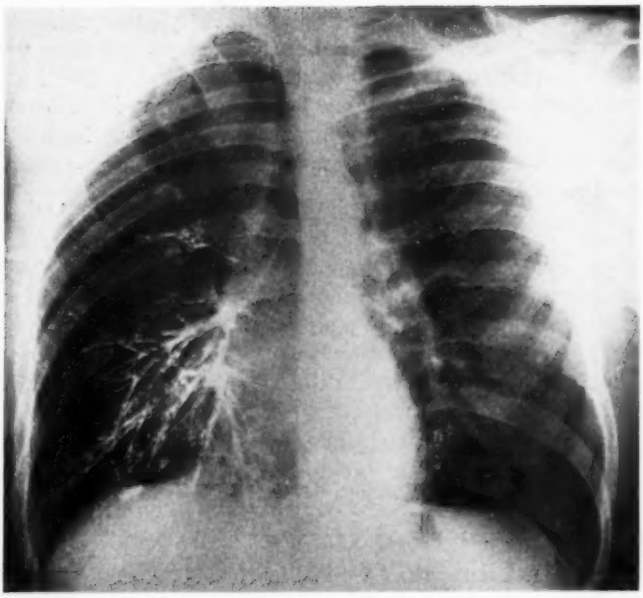


Fig. 3.—PA and lateral roentgenograms of the same patient in Figure 2, made 20 minutes later. The bronchi still show normal sized main trunks abruptly narrowing to a filamentous lumen. These are widely spaced with no alveolar filling.

The bronchoscopic examination revealed moderate hyperemia of the tracheobronchial tree.

The bronchograms revealed no evidence of bronchiectasis. There was bronchial narrowing and attenuation.

Comment: It is our feeling that physiological alterations in the tracheobronchial tree are demonstrable by bronchography. We believe that bronchograms can serve as a source of physiological information in addition to the anatomical demonstration of the bronchial tree. Our experience leads us to believe that bronchial attenuation occurs in a variety of pulmonary lesions and in many instances is indicative of physiological disturbances. This has been observed in apparently normal segments of the lung in patients with bronchiectasis, tuberculosis and chronic lung abscess. Such attenuation is invariably present in asthma and seems to be an essential component of emphysema.

Early in asthma and emphysema the attenuation seems to be reversible, however if present long enough it may become irreversible. Di Renzo seems to regard this attenuation as an invariable forerunner of permanent emphysema. We are not this certain about the pathologic physiology of attenuation and are following a group of patients with attenuation only with the hope that we may learn more of its significance. So far none of the patients followed have developed permanent emphysematous changes.

3400 Spruce St.

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DISCUSSIONS

DR. ALBERT H. ANDREWS, JR. (Chicago, Ill.): The closing remarks on pulmonary function studies came somewhat as a surprise to me, because I think I have seen cases of this type in which there have been alterations in the pulmonary function studies. The actual ventilatory changes—in volume of respiration and efficiency of respiration—may not be altered, but I thought I had consistently observed alterations in the vital capacity curve. As you all know, functional bronchial obstructions are so frequently manifested by a marked curve in the vital capacity, with a slowing of the speed of expiration, producing a fairly characteristic curve. In cases of this type I had thought that the respiratory curve was more of a straight line.

We know how, in heart disease, the respiratory curve is a rather vertical, straight line. Asthmas and emphysemas have a definite curve. In this type of curve I thought it was more of a slanting type of curve, and not so much of an actual change in the shape.

It was interesting to hear the statement made that small amounts of oil are injected during bronchography. In my experience with bronchography, I think this is a point that is well worth emphasizing—that by accurate positioning of small amounts of the oil, and then the accurate positioning of the patient, better delineation is obtained.

The use of spot films is, of course, something that I feel is definitely of importance.

I have been wondering just what bronchial dilator drug was used, and whether any studies were made in relation to the use of atropine in the bronchography.

DR. FRANCIS W. DAVISON (Danville, Pa.): I thoroughly enjoyed Dr. Atkins' presentation, and his beautiful bronchograms.

I think the diffuse narrowing described by Dr. Atkins could be due to edema, resulting from bacterial allergy. Dr. Atkins wasn't specific about the type of secretion, the presence of eosinophiles or about the bacterial flora, but he mentioned that bronchodilating drugs were not effective in changing the pattern. I think that is because the narrowing was probably due to submucosal infiltration.

DR. JOSEPH P. ATKINS (Philadelphia, Pa.): I think, Dr. Andrews, that the problem of pulmonary function is one which we must leave open at the moment. We have seen patients in whom we expected to find a reduction of the maximum breathing capacity—because of reduction of the lumen, but we haven't always found it. That is the reason we have to say at this point that our studies are not uniform enough to be conclusive.

The bronchial dilators which we have used have been adrenalin and aminophylline. Most of these patients have had atropine before the bronchogram was started; as part of their routine pre-medication.

I think Dr. Davison's suggestion that we may be dealing with a bacterial allergy is a very definite possibility.

I think bronchology has come a long way by solving anatomic and mechanical problems. We are coming to a place now where much greater stress must be placed on physiologic and functional problems.

LXXV

THE RATIONAL USE OF ENDOSCOPY IN SURGERY

FREDERICK FITZHERBERT BOYCE, M.D.

NEW ORLEANS, LA.

I speak to you today, by your kind invitation, as a general surgeon with a special interest in thoracic surgery and with a large stake in your specialty, in which I am not myself trained. I am in full agreement with the generous concept which many of you hold, that the field is large enough for all who are willing to devote the time and effort necessary to perfect themselves in it. Obviously, the thoracic surgeon who is able to do his own endoscopic work is, in many respects, in a preferred position. On the other hand, most general surgeons, like myself, even if they had the time, would probably not practise endoscopy often enough to develop maximum skill in it.

My own feeling, therefore, is that the general surgeon who does thoracic surgery would do better to establish a close liaison with the men trained in the endoscopic field. I have had, in the course of my surgical practice, many reasons to appreciate the help and counsel of these associates. I suggest that any surgeon who is not receiving from them similar aid and comfort is not making the fullest possible use of their abilities. It may be that he is not doing his own part, such as letting only the most serious reasons keep him from being present at all endoscopic examinations and making his own inspection of the visualized bronchus or esophagus. The recollection of what he saw, and the immediate discussion of the findings, will be far more useful to him at operation than any mere written report, however complete it may be.

It is a serious error for the surgeon to regard the endoscopist merely as a technician, though he must possess a high degree of technical skill. Nor must the surgeon look upon the endoscopist as a diagnostician. Diagnosis remains the surgeon's ultimate responsibility. Incidentally, that is a point of view that might well be emphasized to many of the recent graduates in medicine now serving as internes and residents. The endoscopist should, instead, be regarded

From the Department of Surgery, Tulane University of Louisiana School of Medicine.

as a consultant in the fullest sense of the term. Only when he is consulted, just as the internist, the radiologist and other specialists, will the patient's best interests be served.

For my own part, I so regard him, and I have only one specification, that he should be surgically minded. A major portion of endoscopic work is concerned with malignant disease, and the only cure for most malignant disease, in the present state of our knowledge, is radical surgery. I shall, in fact, devote the major portion of these remarks to one special form of malignancy, carcinoma of the lung, spending no great amount of time on the other conditions for which endoscopy is useful.

ESOPHAGOSCOPY

Jackson and Jackson[1] have well expressed the rationale of esophagoscopy. Deductive methods of diagnosis, they pointed out, which are unreliable in any event, have no place whatever in the diagnosis of disease of a viscus so easily and so safely inspected as the esophagus. The bougie supplies only inferential information, and the x-ray, useful as it is, cannot be accepted if the findings are negative in the face of clinical evidence of disease.

Esophagoscopy is indicated in any patient who presents any evidence of disordered esophageal function, no matter how slight it may be. Until we follow that principle, carcinoma of the esophagus will remain in its present unhappy state. Brilliant methods of treatment have been devised for it, but close to 100 per cent of the patients with this disease are still dying from it. Esophagoscopy combined with roentgenologic examination and carried out when the first indications of abnormality become evident could promptly improve that picture. The uniform assumption should be that any adult with symptoms referable to the esophagus has some disease which requires treatment and that the chances are that the disease is cancer. In the absence of direct proof, secured by esophagoscopy, that no other disease exists, it is particularly dangerous to assume that he may be suffering from cardiospasm.

I do not agree with the physicians who regard bleeding which is presumably from esophageal varices as a contraindication to esophagoscopy. On the contrary, I believe that this procedure is indicated if there is any doubt at all as to the origin of the hemorrhage. It has its hazards, it is true, but in skilled hands they are not too great to make the differentiation between bleeding of this origin and bleeding from the stomach. The patient, in fact, may lose his life if the differential diagnosis is not made. Gastric bleeding is more

and more regarded as an indication for laparotomy. Bleeding from esophageal varices is preferably treated by the double balloon, with later injection of some sclerosing solution, such as sodium morrhuate. If the balloon is not effective, transesophageal fixation of the bleeding vessels is required. Neither gastric nor esophageal bleeding, however, can be treated definitively until the precise diagnosis is established, and that, I believe, can be best accomplished by a prompt resort to esophagoscopy in any doubtful case.

BRONCHOSCOPY IN PREOPERATIVE AND POSTOPERATIVE MANAGEMENT

Let me begin my remarks on bronchoscopy with a personal experience:

In 1943 I operated on an elderly, obese man with a ruptured appendix. This was in the pre-antibiotic days. He did not tolerate constant intestinal decompression well and he had a disturbing degree of distention for a number of days after operation. He also had an irritating cough, though the internist reported that his chest was clear. On the twelfth day the retention sutures were removed, and several hours later the wound ruptured and the intestines presented in the incision. The abdomen was closed immediately, under very light gas anesthesia. The procedure had scarcely been concluded when there developed, precipitately, the most extreme example of massive atelectasis which I have ever seen. Catheter aspiration of the trachea produced no results. Oxygen was equally ineffective. A bronchoscopist, who was fortunately in the hospital, was summoned and introduced his instrument. Within ten minutes the patient was brought back from the gates of death, quite as promptly and as dramatically as he had gone close to them. He owes his life not to me but to the bronchoscopist who rescued him from suffocation.

We have all of us learned a great deal more about postoperative atelectasis than we knew ten years ago, when this experience occurred. For one thing, we are more aware of the importance of eliminating, before operation, material in the chest which could cause bronchial plugs after operation. This is a particularly essential precaution in all resectional thoracic surgery. Some authorities believe that bronchoscopy should be used routinely as a preoperative measure before all such operations. My own practice is to employ postural drainage before surgery for bronchiectasis or lung abscess but to resort to bronchoscopy without delay if the simpler measure is not promptly effective. Bronchoscopy is clearly indicated, under these

circumstances, before a procedure such as lobectomy, in which spillage of secretions into the contralateral lung might initiate an infection in intact tissue, or thoracoplasty, in which retention of secretions in a compressed lung might easily prove fatal.

Bronchoscopy should be carried out immediately if the patient has vomited on the operating table. This accident is likely to happen only in emergency surgery, in an improperly prepared patient, but the pulmonary gangrene which may follow the aspiration of vomitus into the lung is so dangerous that the mere suspicion that it has occurred is an indication for the use of the bronchoscope. Obvious vomiting, as Culver, Makel and Beecher[2] have shown, is only half of the problem. The silent aspiration of gastric contents by the deeply anesthetized patient is another, unrealized, danger. In a study of 300 unselected surgical patients under general anesthesia, they found that 26.3 per cent had regurgitated gastric contents in the course of operation and that 16.3 per cent had aspirated gastric contents. Their observations suggest a more general use of bronchoscopy after operation than has heretofore been thought necessary.

Atelectasis is unlikely to occur after operation if there is careful attention to such commonsense precautions as aerating the lungs after anesthesia, changing the patient's position in bed until he is able to move himself, early ambulation, and deep breathing and frequent and forceful coughing, under supervision. If there is any suspicion that the cough is ineffectual and that secretions are being retained in the tracheobronchial tree, catheter aspiration of the bronchi, as suggested by Haight and Ransom[3] in 1941, is usually effective. Their advice is sound, however, that if there is any difficulty in introducing the catheter, bronchoscopy will be less disturbing than repeated attempts at catheterization, especially to the seriously ill patient. The chief advantage of bronchoscopy over catheterization is that it is more thorough. Both stem bronchi can be aspirated with certainty under direct vision, and mucous plugs blocking the orifices of branch bronchi are unlikely to be overlooked.

It is a serious error to delay bronchoscopy when there is evidence that any degree of atelectasis is present. Even the patchy variety, with indefinite roentgenologic findings, may go on to pneumonia if it is not promptly checked. As Lahey[4] has emphasized, bronchoscopic aspiration should be regarded as an emergency procedure in the presence of rises in the temperature and pulse, caused, presumably, by accumulations of mucus which cannot be dislodged by catheter suction, forced coughing, or vigorously pounding the patient on the back. Another reason for prompt resort to broncho-

scopy is that if the obstruction has existed for any length of time, the sudden loosening of large amounts of retained secretion could flood a normal bronchus, with possibly fatal consequences.

Although, as will be pointed out later, bronchoscopy is a procedure to be avoided for diagnostic purposes in patients in poor condition, this does not hold for bronchoscopic aspiration either before or after operation. Then it is strongly indicated for weak, exhausted and comatose patients, who do not have the strength to cough up their own secretions. The more critical their condition, in fact, the more urgent is their need for this adjunct measure.

BRONCHOSCOPY IN NON-MALIGNANT DISEASE OF THE CHEST

Bronchiectasis. Bronchoscopy is an extremely useful diagnostic measure in bronchiectasis. It should always be used preliminary to bronchography, which is essential to diagnosis. It is, as already pointed out, a useful preoperative measure in clearing the chest of secretions when postural drainage has not accomplished this purpose completely. In the occasional early case it may be therapeutically successful, if at the same time there is efficient treatment of whatever focus of infection there may be in the upper respiratory tract, or the tonsils, sinuses or teeth. If the bronchiectasis has been caused by a foreign body, and if the process is not too extensive, removal of the object through the bronchoscope will often effect a cure.

With these exceptions, however, bronchoscopy should be regarded as a diagnostic and adjunct measure in bronchiectasis, not as a method of treatment. The changes in this disease are irreversible. It is, furthermore, a disease of recurrences. The fact that a patient is quite well at the moment does not protect him from an exsanguinating series of hemoptyses or a fatal attack of pneumonia next week or next month. Bronchiectasis of any but the mildest degree is attended with such morbidity and mortality under medical management that the physician who advises against surgery for it is assuming a heavy responsibility.

Overholt and Walker[5] have recently made an interesting contribution to the management of bronchiectasis in pointing out that the disease may exist in a hidden or unsuspected form in the asthmatic patient. Bronchial obstruction is the principal cause of the clinical manifestations of bronchial asthma, though whether asthma and bronchospasm precede bronchiectasis, or vice-versa, it is not possible to say. If bronchoscopy is repeatedly employed in such

cases, sometimes under deep ether, it removes thick, tenacious and adhesive secretions and indicates the location of bronchostenosis, extrinsic pressure and other abnormalities. Bronchoscopy must be followed by bronchography, for precise localization of the bronchiectatic areas. Overholt and Walker's report is illuminating: Of 76 patients with severe bronchial asthma whom they observed over a four-year period, only 4 had previously been examined in this manner, and 3 of the 4 examinations were unsatisfactory. The fourth patient was found to have bronchiectasis as well as asthma, and the same combination was demonstrated in 27 of the other 75 patients when they were properly examined.

Lung Abscess. The first point to be made about lung abscess is that at the present time the most frequent underlying cause is carcinoma, which must be thought of first of all in all adult patients. This possibility is one very sound reason for not regarding bronchoscopy as a definitive method of treatment in this condition. The other reason is the increasing perfection and safety of thoracic surgery.

Most thoracic surgeons are now agreed that the majority of lung abscesses become chronic because they are not interrupted in the acute stage. In other words, acute lung abscess is usually a surgical disease, provided, of course, that one is fortunate enough to see the patient sufficiently early to drain the abscess promptly. Such measures as rest, fresh air, antibiotic therapy, postural drainage and bronchoscopic drainage should be employed, of course, before operation is resorted to, and it may be that they will be curative. The decision as to whether they should be continued depends not upon how long they have been used but upon what they are accomplishing. The only justification for their continued use is improvement in the patient's general condition, associated with radiologic evidence of improvement. Deterioration in his condition, with radiologic evidence of progression or with stationary radiologic findings, is an indication of failure of conservative therapy and of the urgent need for surgical drainage.

In the chronic stage, bronchoscopy has nothing therapeutic to offer the patient with a lung abscess except, perhaps, temporary palliation. When this stage is reached, only surgery, in the form of lobectomy or pneumonectomy, holds out a prospect for cure.

Tuberculosis. Before surgery is undertaken in tuberculosis, it is necessary to know whether stenosis of any of the major bronchi is present or whether endobronchial extension has occurred. Either of these conditions is an indication for resection, but the decision can be made only after bronchoscopic examination.

TABLE 1.
BRONCHOSCOPIC OBSERVATIONS IN 132 OF 235 FATALITIES FROM BRONCHOGENIC CARCINOMA.

77	positive (59 with biopsy confirmation)
2	suggestive
5	incomplete
2	inconclusive
30	negative
16	reported as tuberculosis, endotracheal disease, bronchiolitis, external compression, benign tumor, mass of undetermined origin, purulent exudation, bronchoesophageal fistula, carcinoma of the larynx, trachea and esophagus.

BRONCHOSCOPY IN CARCINOMA OF THE LUNG

The widest field of usefulness for bronchoscopy today, as well as the most urgent indication for its use, is in the diagnosis of carcinoma of the lung. This is a disease which is steadily increasing in incidence. We know exactly what to do to cure it. Yet in the hands of the best thoracic surgeons the five-year salvage amounts to barely five per cent.

I have recently had the depressing experience of studying 235 fatalities from carcinoma of the lung which occurred at Charity Hospital of Louisiana at New Orleans between 1947 and 1952 inclusive.[6] Only 38 of these patients had been submitted to pneumonectomy, which was palliative in 34 cases, and only 24 others had been submitted to exploration. In the light of this analysis, and of my personal experience in private practice, I doubt that we are making the fullest possible use of bronchoscopy in this disease. I make that statement with full awareness of the fact that a third or more of all lung cancers are so situated that they cannot be reached by the biopsy forceps.

One hundred thirty-two of these 235 patients were studied by bronchoscopy (Table 1). It was omitted in the remaining 103 cases for a variety of reasons: Some patients died too promptly after hospitalization to permit a complete work-up. In some instances, often with a great deal of justification, the disease was not suspected before postmortem. In other instances there was also no suspicion of the disease, though in retrospect there seems little excuse for the error.

Let us examine the implications of these bronchoscopic findings (Table 1). In the 16 cases in which diagnoses other than cancer were made, only 3 patients were submitted to exploration and only 1 to pneumonectomy, which was delayed for a year; at the end of that time a second bronchoscopy showed that the purulent exudate reported at the first examination originated in a carcinomatous lung abscess. A number of the patients were hopelessly ill when they were examined and nothing could be done for them. But others were under observation for fairly long periods of time, and a more aggressive attitude in the face of obvious abnormalities, whether or not carcinoma was diagnosed, might have changed the course of events.

When bronchoscopic biopsy is positive, the diagnosis is established. I wonder, however, whether we are not relying too much upon the results of biopsy and paying too little attention, as the observations just cited suggest, to indirect evidence of disease. We could well take the position that even when a tumor is not visualized, and even when tissue is not available for biopsy or material is not secured for cytologic examination, indirect evidence of bronchial abnormalities should be given a great deal of weight. Unless stenosis, hyperemia, deformity, exudation, rigidity, infiltration and similar changes can be promptly explained by nonsurgical diagnostic methods, they should be regarded as indications for prompt exploration of the chest. Compression atelectasis is a manifestation of many bronchogenic carcinomas. Narrowing of the lumen of the bronchus, fixation, and widening and distortion of the carina may be caused by other diseases, such as an old tuberculosis, but it is a safe plan to assume that they are caused by malignant disease and to resort to thoracotomy without delay to confirm or exclude the diagnosis.

Negative bronchoscopic findings in clinically suspicious cases should be accepted with the utmost reserve, if at all, unless roentgenograms have previously shown the tumor to be located in an inaccessible area. Then the cytologic study of secretions or of bronchial washings secured at bronchoscopy may be useful, but again negative results should be accepted with reserve. Some of the patients in the series studied at the New Orleans Charity Hospital lost their lives because negative findings—physical, radiologic and cytologic as well as bronchoscopic—were accepted as definitive. It is only fair to add, however, that pneumonectomy was carried out in 13 cases and exploration in 8 cases in the face of negative bronchoscopic reports.

One particularly serious diagnostic error was apparent in this series, the repetition of bronchoscopy and other examinations over a period of weeks if the first examination was not conclusive or was negative. In a disease such as carcinoma of the lung there is no excuse for leisurely observation. The time for salvation is always immediately at hand. Diagnosis is a matter of extreme urgency. All tests should be carried out promptly, preferably concurrently, if not, one immediately after the other. An incontrovertible diagnosis, secured by repeated tests at various intervals of time, may seal the patient's doom. If, within a period of two weeks or thereabouts, a positive diagnosis cannot be made, or bronchogenic carcinoma cannot be positively excluded, then exploration of the chest should be undertaken, with no additional delay. This concept is neither radical nor reckless. Instead, it is life-saving. In this disease conservatism is fatal.

In these 235 patients there were 84 who consulted physicians before they entered the hospital. Thirteen were advised to go to the hospital at once. The other 71 were treated for various conditions, or on no diagnosis at all, for periods ranging up to two years. Half a dozen were submitted to radiologic examination, but not a single one was bronchoscoped before he was hospitalized. Clearly, some way must be found to educate physicians who do not specialize in diseases of the chest to two facts: The first is that carcinoma of the lung is a disease of increasing frequency. The second is that prompt roentgenologic and bronchoscopic examinations are the most readily available methods of diagnosing it.

The work of Rigler and his associates[7] on early x-ray diagnosis is one of the really important recent advances in carcinoma of the lung. X-ray examination is not supposed to be useful in the incipient stages of the disease, but, as these observers have shown, the evidence is there if it is only sought for carefully enough. In a group of 50 patients with proved carcinoma of the lung they were able to show, on films taken at varying periods of time before there was clinical evidence of the disease, that in every instance there was some radiologic abnormality, overlooked at the time but full of significance in the light of after events. Rigler and his group were further able to show, from these observations, that the duration of life in carcinoma of the lung is, on the average, ten months longer than it has previously been considered to be. These ten months, as they say, represent many new opportunities for salvage in this disease. A more careful interpretation of roentgenograms, a more liberal interpretation of bronchoscopic observations, even if they

are not pathologically positive for cancer, and a readier resort to exploration of the chest on suspicion alone would soon change the outlook in carcinoma of the lung for the better.

CONTRAINDICATIONS TO BRONCHOSCOPY

Let me say, in conclusion, that I regard advanced cardiovascular disease and aortic aneurysm as the only absolute contraindications to bronchoscopy. On the other hand, I do not believe that there is ever any justification for this measure in hopelessly ill patients, merely to complete a planned diagnostic routine. In the 235 fatal cases studied at the New Orleans Charity Hospital, 4 patients died within four to thirty hours after they had been bronchoscoped. Two went into shock during the procedure and required tracheotomy. All 4 were close to death, and the fatalities could not possibly be charged to the bronchoscopic examination, though undoubtedly all were hastened by it. The point is, however, no matter how skillfully the examinations were performed, that these patients were put to an ordeal to which they should not have been submitted. The satisfaction of a positive bronchoscopic examination or biopsy in such cases as these is entirely academic, and I believe most bronchoscopists will agree with me that it is not worth what it costs.

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LXXVI

THE CHANGING CONCEPT OF TUBERCULOUS TRACHEOBRONCHITIS

JOHN J. O'KEEFE, M.D.

PHILADELPHIA, PA.

The problem of endobronchial tuberculosis has long been appreciated as being one of disagreeable consequence. The condition of inflammatory reaction of the bronchial mucosa, with its attendant edema, ulcerations, granulations, and consequent stenoses, all acting as obstructive phenomena, has caused prolongation of the course of the disease, interfered with the proper execution of collapse therapy, and eventuated in such unwanted complications as atelectasis, bronchiectasis, and the ballooning of cavities.

Fully appreciating the seriousness of tuberculous endobronchial lesions, it seems only fitting that in the initial appraisal of the tuberculous patient, appropriate consideration be tendered the condition of the bronchial mucosa. It is of equal importance that direct examinations of the bronchi be made during the course of management of the tuberculous patient. Such calamities, as above mentioned, are the direct consequence of failure to carry out these examinations.

Bronchoscopy, as it is applied to the tuberculous patient, cannot be substituted for by any other method of examination. Although it may be variously indicated, it is aptly paraphrased to state that "the indications for bronchoscopy consist in the presence of bronchopulmonary symptoms, the explanation for which is incomplete or unsatisfactory."[1] Specifically, however, in tuberculous bronchitis, the role of bronchoscopy is a complex one: it is a means by which etiologic diagnoses may be obtained; it is a means by which the progress of the disease may be observed directly; and at times, it is a means of therapy.

As a diagnostic method the indications for bronchoscopy are well defined. It is of particular merit when used to secure secretions from a section of lung indicted as being suspiciously tuberculous by physical examination or by x-ray study; it is a singular means for ascertaining the cause of bronchial obstruction in a suspected or known tuberculous patient; often times it is used to identify the

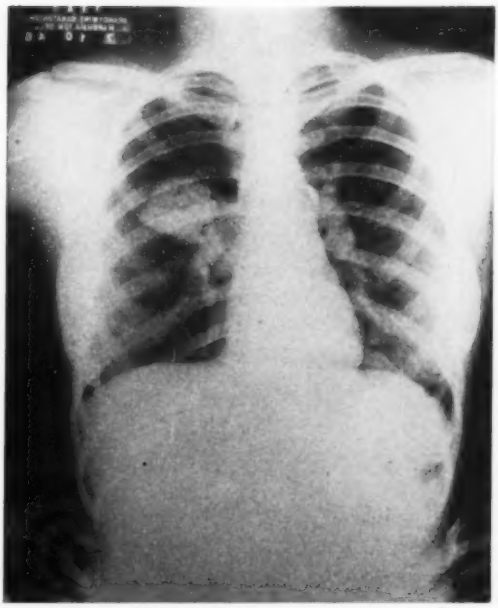

Fig. 1.—Roentgenogram illustrating the presence of atelectasis of the right upper lobe following pneumothorax.

source of hemoptysis; and it is indispensable in differentiating the presence of other broncho-pulmonary lesions coexisting with tuberculosis. As a correllary to this discussion it is interesting to note the ever mounting number of instances recorded in the literature of bronchogenic cancer coexisting with tuberculosis, diagnosed through the practice of "routine" bronchoscopy in tuberculous patients.

During the course of management of the tuberculous patient, several situations occur in which the bronchologist is called upon to report on the condition of the bronchial mucosa. Such examinations are indicated in anticipation of collapse therapy, in the evaluation of response to therapy of previously ascertained bronchial lesions, and in the evaluation of the effects of collapse therapy.

Soon after 1946, when streptomycin was first accepted as a suppressive influence on certain forms of pulmonary tuberculosis, it was likewise appreciated that this same antibiotic had a remark-

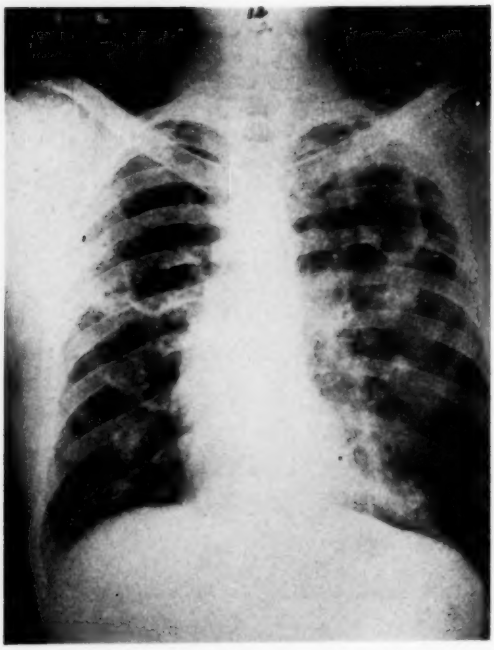

Fig. 2.—Roentgenogram of a giant cavity of the left upper lobe which failed to collapse following pneumothorax.

able way of resolving tuberculous endobronchial lesions.[2] Further, this antibiotic agent effected such resolution in a relatively short period of time and without the formation of secondary stenoses. Since that time, through continued clinical research, refinements in administration have been made, and more effective application of the drug has been developed. During these years of clinical investigation, it has been also shown that the various methods of administration of streptomycin have particular indications: that streptomycin, when administered parenterally, in sufficient dosage, is effective on endobronchial lesions, as well as on the pulmonary lesions, is an accepted fact; that aerosol administration alone is insufficient medication, though not ineffective; and that the most *rapid* repair of tuberculous endobronchial lesions is attained through the concomitant parenteral-aerosol method of administration. Current opinion also holds that in order to maintain these effects, there must be continued suppres-

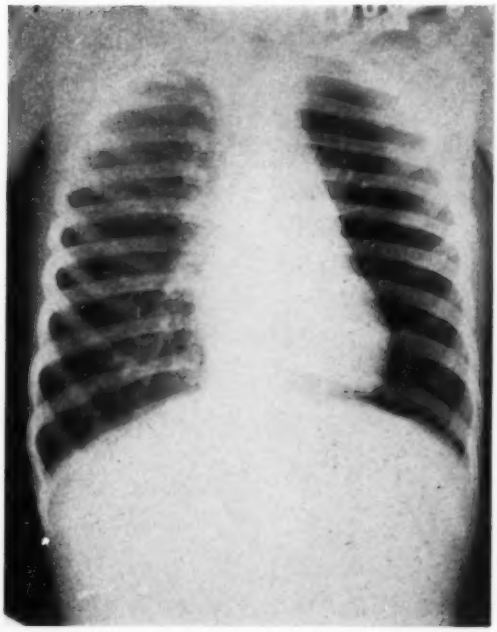

Fig. 3.—Roentgenogram illustrating the presence of obstructive emphysema of the left lung. Bronchoscopy revealed the presence of a pedunculated tumor in the left main bronchus. Histologic report: tuberculoma.

sion of the dissemination of infected secretions from the parenchymal lesion.

Today, the situation that pertains concerning the position of tuberculous endobronchial disease, in the overall management of the tuberculous patient, is in every respect dependent upon the application of this knowledge of therapy. The incorporation of antimicrobial agents in the management of the tuberculous patient has reduced the incidence of obstructive endobronchial disease to such a point that it is now only an occasional patient who presents bronchoscopic or roentgenographic evidence of such lesions. It is significant[3] to note that this condition parallels that of tuberculous laryngitis, the incidence of which is currently reported by American phthisiologists as being between two to four per cent of all cases.

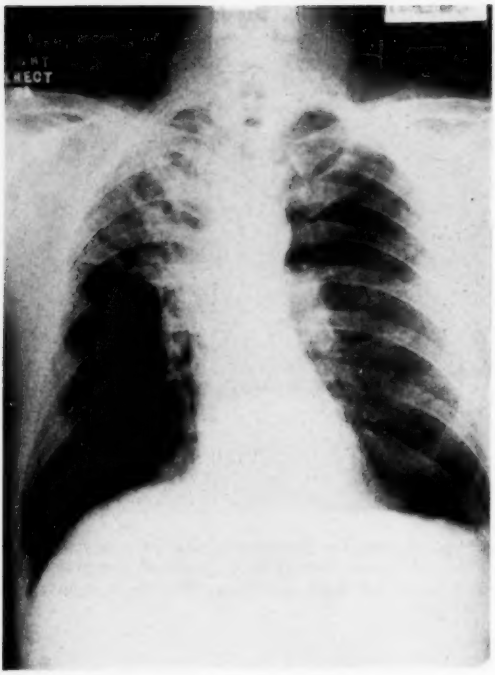

Fig. 4*a*.—Roentgenogram illustrating the presence of a marked infiltrative reaction containing multiple areas of radiolucency involving the right upper lobe. Roentgen interpretation: fibroid tuberculosis with multiple cavitation.

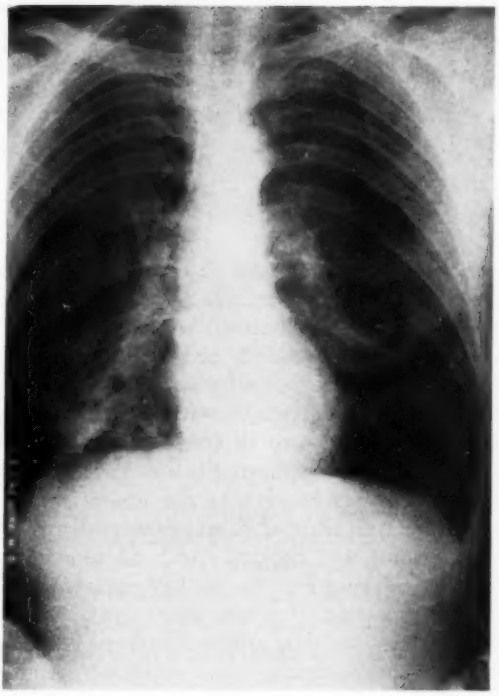

Fig. 4*b*.—Roentgenogram illustrating the presence of infiltrative reaction involving the right lower lobe in the instance of a patient who was reported to have had a positive sputum for acid-fast bacilli. In both these instances the diagnosis of carcinoma of the lung was obtained through bronchoscopic examination.

TABLE 1.—RESULTS OF STREPTOMYCIN THERAPY IN TUBERCULOUS TRACHEOBRONCHITIS.

	PATIENTS SELECTED BRONCHOSCOPICALLY			
	CONCOMITANT PARENTERAL AND AEROSOL	PARENTERAL ALONE		AEROSOL ALONE
		2 GM/DAY	1 GM/DAY	
Healed	9	4	5	1
Improved	1	2	9	6
Unimproved	0	0	4	3

And now, having obtained this objective—the reduction of the incidence of tuberculous endobronchial disease to such a state that its occurrence is aptly termed "unusual"—we are today confronted with two situations that are equally as serious in their consequences and somewhat more difficult to treat successfully. The first of these refers to that group of individuals who persistently harbor endobronchial lesions unresponsive to all forms of antimicrobial therapy. That such a development was inevitable was known and appreciated even in the initial reports concerning the limitations in the use of streptomycin in the treatment of tuberculous patients. As reported previously,[2] concerning the subject under discussion, it was stated that "of equal consequence but less well appreciated is the development of streptomycin-resistant strains occurring in instances of prolonged administration . . . this, rather than being an acquired trait, is the survival and multiplication of a small number of inherently resistant organisms." The presence of this complication, as it exists today, undoubtedly is the result of our own efforts: however, there is sufficient reason to believe that control of these resistant strains will eventually occur with the development of better or more effective antimicrobial agents.

Secondly, and of equal seriousness, there are now noted increasing numbers of tuberculous patients who present signs of bronchial obstruction due to the compressive effects of extrabronchial tuberculous lymph nodes. This is an equally critical situation, for, when of sufficient size, these masses cause obstruction just as effectively as do the endobronchial lesions. In addition, these nodes further complicate this situation in their predisposition to perforate into the bronchial lumen. The complications of such obstructions appear

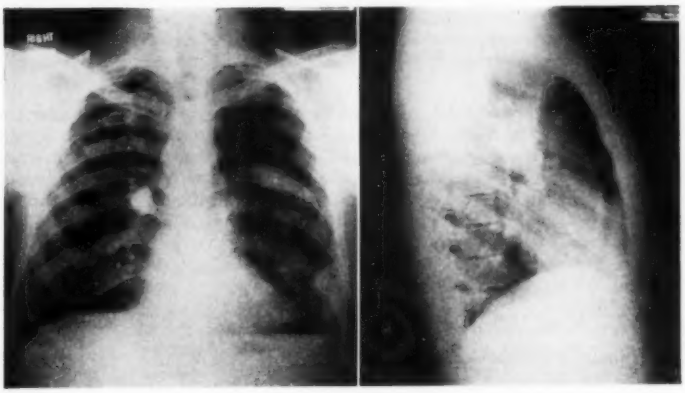

Fig. 5 a and b.—PA and lateral roentgenograms illustrating the presence of dense hilar lymph nodes, one of which is seen encroaching on the lumen of the right main bronchus.

to be poorly responsive to medical therapy and consequently, in many instances, necessarily eventuate in some form of surgical excision.

In summary then, it seems somewhat evident from the foregoing discussion that the bronchoscopic aspect of tuberculous tracheobronchitis has changed materially since the incorporation of antibiotic and antimicrobial agents in the treatment of pulmonary tuberculosis. That such changes have been for the good of these tuberculous patients is certainly unquestioned. The development of more effective therapeutic agents has in no way detracted from the value of, nor allowed for the elimination of bronchoscopy as an integral component in the management of these patients. Conversely, it has repeatedly been shown that it is only through the practice of bronchoscopy that the complications of bronchial obstruction may be averted or that other bronchopulmonary disease coexisting with tuberculosis may be properly identified.

255 SOUTH 17TH STREET.

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DISCUSSIONS

Dr. Urpo Siirala (Turku, Finland): Seventy-five tuberculous patients were subjected to bronchoscopy at the Kiljava Sanatorium, Finland, for diagnostic and therapeutic purposes during 1945-1948. A part of the observations made will be reported.

In 10 out of 17 unilateral pneumothorax patients who were still under treatment, and whose expectorations contained bacilli, the active focus of disease was found in the untreated lung, from which infected mucus exuded into the bronchi (See Table I). In these cases the changes in the bronchi were comparatively slight. On the other hand, severe tuberculosis was found in the bronchi of the lungs treated with collapse in 7 patients, in which the pulmonary process was still active in the collapsed lung, in spite of the pneumothorax therapy being applied.

TABLE I.
COLLAPSE TREATMENT AND TUBERCULOUS BRONCHITIS.

Unilateral pneumothorax	17 cases
10 patients: active focus in previously untreated lung. Relatively slight changes in the bronchi.	
7 patients: active focus in lung treated with collapse. Marked bronchial changes on diseased side in all cases.	
Unilateral pneumolysis and thoracoplasty	11 cases
9 patients: active process in both lungs. More distinct bronchial changes on diseased side operated on.	
9 patients: active focus in collapse-treated lung. Marked change in bronchus of diseased lung in all cases.	

Active processes in both lungs were found in only 2 out of 11 cases treated with unilateral pneumolysis or thoracoplasty. There were more distinct bronchial changes on the side operated upon. In 9, the active focus was found in the lung treated with collapse, and in all cases there were distinct tuberculous changes in the bronchus of the diseased lung.

The conclusion to be drawn from the above investigation is that infection of the bronchi may easily result if the collapse of the lung is not effective and the lung still secretes bacteria-containing mucus into the bronchi. This is probably due to several causes—for instance, stagnation of infected mucus in the bronchi of the collapsed lung, and the unnatural physiological conditions which may decrease the power of resistance of the bronchial mucosa.

Dr. Ricardo Tapia Acuna (Mexico City, Mexico D. F.): I think Dr. O'Keefe has pointed out very well the problem of tuberculous bronchitis. I just want to stress some matters about it.

I remember seeing a case in the Tuberculosis Sanatorium of Mexico, where the patient had been interned in the hospital for more than two months. He was not improved. Bronchoscopy was asked by his doctor, so I examined him with a bronchoscope, and found a big carcinoma. That means that we should bronchoscope these patients if they are not responding to treatment.

I believe that cases of bronchial complications have decreased since we began using streptomycin. However, we have also seen cases of resistance as Dr. O'Keefe said, especially in hyperplastic bronchitis.

I still believe that it is not a bad idea to do bronchoscopy as a routine procedure in the examniation of all tuberculous patients—with some exceptions, of course.

DR. PAUL H. HOLINGER (Chicago, Ill.): I would like to pay particular attention to the last part of Dr. O'Keefe's paper. Certainly, bronchial tuberculosis has changed in the last few years, due in part, to the advent of the chemotherapy used; but also I believe to the decreasing incidence of pneumothorax. As Dr. Siirala mentioned, there is a relationship between collapse therapy and the presence of bronchial tuberculosis.

We now rarely see the conventional textbook picture of the exudative, ulcerative type of lesion, but more frequently see the lesion which is due to perforation or obstruction of the bronchus by a node, a lesion formerly associated more frequently with tuberculosis in children. I think these phenomena are being seen with increasing frequency in adults. Recent papers by Iselin in Switzerland have brought this very closely to our attention. They point out that the tuberculous pneumonias and tuberculous exacerbations which were contraindications, formerly, to bronchoscopy, are now very strong indications for bronchoscopic aspirations of these areas, because so many of the tuberculous pneumonias are apparently developed on the basis of a rupture of the tuberculous node into the bronchus itself. So the aspiration even in the acutely ill patient, with an increasing tuberculous pneumonic process may be indicated, whereas we formerly thought that procedure was highly contraindicated.

Furthermore, it is suggested that in addition to the use of antibiotics, we should return to the use of silver nitrate in the management of these patients.

DR. H. JAMES HARA (Los Angeles, Calif.): Some of you know that I was in Japan last spring, and at that time I had an opportunity to come in contact with a large number of patients with tuberculosis of the bronchus.

Eighty-five per cent of the population of Japan is suffering from tuberculosis. At one of the institutions over there, Hokkaido University, Dr. Jo Ono, who trained under Dr. Clerf, is in charge. There I had the privilege of seeing Dr. Ono's treatment of a patient with tuberculosis of the lower air passage. In addition to giving streptomycin intramuscularly, he applied .5 gm of streptomycin in 1.5 cc of normal saline solution as a local application to the tuberculous lesion. He is reporting good results.

I thought you might be interested in knowing that a topical application of this drug has seemed to be effective—not in all cases, but in some of the cases I had the privilege of seeing.

DR. TAUNO K. PALVA (Turku, Finland): I was very happy to listen to the last speaker, who told us about the same method and report in one case. In my mind it further illustrates the change in treatment and prognosis due to antibiotics, and in that connection I would like to present a case history with some interesting features.

The patient was an 18-year-old white female. In Finland we do not have Negroes or other colored people. She was taken to a tuberculosis sanatorium for a small lesion of the right lung and trachea.

During a few months of conservative therapy, the lung findings disappeared, but the tracheal condition was unchanged. The patient was then discharged from the sanatorium, and was told to go and consult an otolaryngologist for further treatment of the trachea.

A few weeks later she was seen by an otolaryngologist, and the lesion was found to be on and below the subglottic area. This part of the trachea was covered with yellow membrane, and exuded a rather foul-smelling odor.

Treatment by direct application of silver nitrate through the bronchoscope tube was immediately started. In a few weeks, however, it became clear that this treatment would not result in success. The process spread rapidly down the trachea, and the membrane formation involved here increased tremendously.

At this stage the patient was seen in our clinic. She was rather unsocial, already, because of the intolerable smell. The vocal cords were intact, and the lung x-rays showed no active process going on.

On laryngoscopic and bronchoscopic examination, the subglottic area as well as the mucous membrane of the trachea were found to be covered with yellow membrane and granulations down to the coryna.

During a two-weeks period in the hospital, the patient was given 1.5 grams of streptomycin daily, parenterally. In addition to this, with a curved-tip spray, in indirect laryngoscopy, I administered 1 gram of streptomycin solution daily to the subglottic area and the tracheal mucosa. In two weeks most of the crust formations disappeared, there was no more foul-smelling material, and the patient was discharged from the hospital. She was continuously watched in the outpatient clinic for a short period of time, and intramuscular streptomycin injections were discontinued ten days after her discharge from the hospital.

Half a year after cessation of this treatment I saw this patient again. She was in excellent health, but showed a very small yellowish area, about the size of a dime, in the subglottic area. The whole tracheal mucous membrane showed a normal appearance, and there were neither granulations nor membrane formations. The foul smell was also absent.

In this case, the combined local and general adminstration of streptomycin thus proved very effective in the treatment of intratracheal tuberculosis.

DR. JOHN J. O'KEEFE (Philadelphia, Pa.): I sincerely thank the discussers of this paper. It is most gratifying to see that, somehow or other, you have stimulated such an interesting discussion.

I would like to review, for just a second, the two principal purposes for presenting this paper—which I have summarized in my conclusions.

First, it is ever more important now that we be on the lookout for the so-called resistant strains of the tubercle bacillus—resistant to the effects of streptomycin—making sure that we do not limit our therapy to streptomycin in such instances.

The newer drugs, including the iso-nicotinic acid hydrazid types, have a place in the therapy of the tuberculous patient, particularly in those instances wherein the organisms infecting the patient are resistant to streptomycin.

The other point, of course, is that there appears to be a change in the way by which the lymphatics are limiting, or failing to limit, the spread of this disease. The development of large peri-bronchial nodes is a situation very similar to the one we saw many years ago; particularly the type seen in children, characterized by suppurating cervical lymph nodes. It might be said, too, that we are again encountering suppurating tuberculous cervical nodes, not just in children, but in adults with pulmonary tuberculosis, the spread of which is not being controlled by antimicrobial therapy.

LXXVII

A BRONCHOLOGIST'S EXPERIENCES WITH INFECTIOUS ASTHMA

F. W. Davison, M.D.

Danville, Pa.

This paper is based on the study of the hospital charts of 50 patients who had chronic suppurative bronchitis and asthma. Suppurative bronchitis was manifest by purulent sputum. Smears made from the bronchial exudate usually showed a heavy bacterial flora with Gram-positive cocci predominating. Often there were many neutrophils, but many smears also showed large numbers of eosinophils in the endobronchial exudate. Smears must be examined with an oil immersion lens, otherwise the bacterial flora will not be seen. In my opinion the emphasis on eosinophils as an index of "allergy" has caused the infectious factor to be ignored or under-treated. It should be recognised that bacterial allergy causes an outpouring of eosinophils of greater degree than does food or inhalant allergy, as was shown by Cooke.[4, 5]

The best evidence that infection alone can cause asthma is its rapid response to adequate antibiotic therapy. Another point of evidence is the production of acute asthma by too large a dose of autogenous vaccine, just as one can precipitate pollen asthma by too large a dose of pollen extract in a person whose bronchial mucosa is sensitized. Another obvious indication of bacterial allergy is the history that the patient has asthma only at the time of acute respiratory infections.

Quoting A. R. Rich,[12] "It is important to recall that there are two main types of sensitization, which differ from each other in a number of ways. One of these is the tuberculin type of hypersensitivity, so called because the reaction of the tuberculous body to the proteins of the tubercle bacillus is its prototype, though the same form of hypersensitivity develops as a result of infection with other bacteria, fungi and filterable viruses. The other is the anaphylactic type which, too, may result from infection with microorganisms, but also from contact with a wide variety of substances derived from

From the Department of Otolaryngology and Broncho-esophagology, Geisinger Memorial Hospital, Danville, Pa.

other sources. Each type arises from a bodily alteration resulting from parenteral contact with a foreign antigen, or with a non-antigenic substance that is capable of uniting readily with body protein to form an antigen foreign to the host. When this bodily alteration has occurred, tissues will be injured, and even killed, by local contact with amounts of the foreign substance that would be harmless to the normal body; and constitutional symptoms, and even death will occur when amounts that are entirely harmless to the normal body enter the blood stream of the hypersensitive one. . . . "

" . . . We found that while in both types of hypersensitivity extravascular tissue cells can be damaged and killed at the site of injection of the specific antigent, the death of the cells in anaphylactic local reactions is the result of vascular damage that interferes with the nutrition of the cells, whereas in the tuberculin type of hypersensitivity the cells of even an avascular tissue such as the cornea, or, indeed, cells from the sensitized body, washed and isolated in tissue culture, are killed by contact with the specific antigen. . . . "

"The tuberculin type of hypersensitivity can play an important role in the pathogenesis of infection. As a result of the development of this type of sensitization, even microorganisms which possess little toxicity for the normal body become able to damage seriously the tissues of the sensitized one."

ANALYSIS OF DATA

The ages of the patients in this group is indicated in the following table.

TABLE I.

AGE OF PATIENTS BY DECADES.

1 - 10 years	0 cases
11 - 20 "	1 case
21 - 30 "	4 cases
31 - 40 "	5 cases
40 - 50 "	10 cases
51 - 60 "	20 cases
61 - 70 "	10 cases

Thus it is seen that this disease usually afflicts people over 40 years of age as has been pointed out by previous papers by Brown,[3] Forman,[8] and Davison.[6]

TABLE II.

DURATION OF SYMPTOMS.

6 - 12 months	6 cases
1 - 5 years	18 "
6 - 10 years	9 "
11 - 20 years	12 "
21 - 30 years	5 "

This table showing duration of symptoms indicates many years of ineffectual treatment and a large amount of human misery. Many of these patients had five to fifteen hospital admissions.

The infectious factor is the most frequently overlooked and under-treated factor in chronic asthma. I think this is true because of the over-emphasis on eosinophils and because of inadequate bacteriologic study.

Thirty-three of the fifty patients had chronic suppurative and hyperplastic sinusitis. Improvement in the asthma usually paralleled successful treatment of the sinus infection. Most of my errors have been due to inadequate surgical treatment of the coexisting sinus infection.

Four of the fifty had allergic pulmonary consolidation, better known as Loeffler's syndrome. Five had extensive pulmonary fibrosis, so that moderate dyspnea persisted after the broncho-pulmonary infection had been cleared up. Three had bronchiectasis. One had bullous emphysema.

The value of surgical treatment of bronchiectasis in asthmatic patients has been demonstrated by Overholt and Walker.[11]

Forty of the fifty had 70 skin tests made by the skin puncture method described by Walker,[14] plus three intradermal tests with Endo's dust extract using 1-10,000, 1-100,000, and 1-1,000,000 dilutions. Twenty-four of the forty had completely negative reactions. Of the 16 positives, 12 patients had positive reaction only to dust. I suspect that bacterial protein in the dust extract accounted for these positive reactions, because most of them had no clinical sensitivity to dust. Coexisting food or inhalant sensitivity should of course be treated.

Thirty of the fifty patients had bronchoscopic aspirations. Some patients had six to ten bronchoscopies because they had symptomatic relief by removal of the gummy endobronchial exudate. Smears and cultures for the other 20 were made from morning sputum specimens. As our medical treatment gets better, bronchoscopic aspirations becomes necessary less frequently.

TREATMENT METHODS

I find the following measures most essential: aminophyllin 0.5 gm in a litre of 5% glucose given by intravenous drip over a period of 4 hours repeated two or three times a day whenever there is much evidence of bronchospasm. For acute dyspnea I prefer 0.3 cc of 1-1000 aqueous epinephrine subcutaneously every 15 minutes until relieved. I will not treat any patient who insists on using an adrenalin nebulizer. The ischemia produced by this method is harmful to the bronchial epithelium and the long-term damage has been well demonstrated by Benson and Perlman.[2] These investigators studied the records of 2236 cases of asthma and found that 648 of them used epinephrin nebulizers. Forty-eight patients in this group died, a death rate of 5 times greater than the rate in a comparable group of patients who did not use adrenalin nebulizers. Autopsies showed loss of cilia, metaplasia of epithelium from columnar to pseudostratified with desquamation and ulceration.

In most cases, an apparent need for oxygen indicates either neglect, or ignorance of the more useful and effective therapeutic measures.

Long-term effective treatment means eradication of infection in the tracheobronchial tree as well as in all other parts of the body. Tracheobronchial infection can be eradicated by antibiotics provided the germs are sensitive, the dose of antibiotic is large enough, and the treatment is continued long enough. The most frequent organism found was the staphylococcus. Invitro tests showed some strains requiring 10-20 units of penicillin per cc for bactericidal effects. Of eleven patients having cultures showing a staphylococcus *not* sensitive to 5 units of penicillin per cc only one had good results with penicillin therapy. This patient had a bilateral Caldwell-Luc and transantral ethmoidectomy plus cortisone for 7 days. Thirteen patients had cultures showing a staphylococcus sensitive to the 5 unit penicillin plate. Results were good in all 13, but moderate dyspnea on exertion persisted in two who had marked pulmonary fibrosis. Their purulent sputum did not recur.

In this series of 50 cases I used from 3 to 10 million units of fortified procaine penicillin intramuscularly daily for periods of 6-12 days. The patients with sensitive germs did best. The following quotations from Eagle and associates[7] state clearly the reasons for using large doses of penicillin: "The most rapidly effective schedule of penicillin administration is one that continuously provides the maximally effective concentration against a particular organism at the focus of infection."

"Sensitivity tests demonstrate not the concentration of penicillin that kills a particular organism at the fastest possible rate, but rather the concentration that prevents possible growth under the arbitrary conditions of the particular test. The maximally effective (most rapidly bactericidal) concentration is usually three to ten times this barely inhibitory concentration."

"A serum conentration 10 times the sensitivity level will usually provide the maximally effective concentration at the site of infection."

Penicillin aerosol is potentially dangerous, because it may sensitize the bronchial mucosa.

Twenty-eight of the 50 were treated also with cortisone by mouth. The initial daily dose of 200 mg was gradually reduced over a two-week period so that the average total dose per patient was 900 mg. No toxic effects were seen in this group of patients treated with cortisone. Cortisone has been invaluable in rapidly suppressing asthmatic symptoms. It is not curative, it does not kill germs, therefore the physician should attempt to eradicate the infection by other measures. Morphine also relieves asthma, but the asthmatic patient should not become dependent on morphine, adrenalin nebulizers or on cortisone.

The following case report illustrates what can be accomplished by penicillin without cortisone.

Female aged 44. Chief complaint, asthma of one year's duration. She had severe asthma for two months prior to admission. She used an adrenalin nebulizer frequently for a year and almost constantly for two weeks. She had been unable to lie down for two weeks. She took cortisone without any improvement before coming to the hospital. The leukocyte count was 17,300 but her temperature was normal. Brochoscopic aspirations yielded thick gummy grey muco-pus, smears from which showed numerous eosinophils

and neutrophils. The cultures showed staphylococcus aureus and streptococcus viridans, both sensitive to 3 units of penicillin. The treatment used was fortified duracillin 4 million units a day, plus streptomycin 1 gm. daily for one week, plus i.v. aminophyllin for 6 days, and potassium iodide by mouth for a period of 2 weeks. Several infected teeth were removed. The skin tests were all negative. She weighed 82 lbs. on admission. She was symptom free by the 9th day. Thirteen months later she said her health was good, she had had no recurrence of asthma and had gained 30 lbs. in weight.

Large doses of penicillin plus adequate sinus surgery have given me better results than any other long-term program of treatment. Effective treatment means elimination of all chronic foci of infection.

For highly penicillin-resistant staphylococcus infections, autogenous vaccines are helpful. I must confess only thirteen patients in this series had vaccine therapy. The value of vaccines has been stressed by Benson,[1] Guerrant,[10] Grorud,[9] Thomas,[13] and many others. For its historic interest, I quote the following from Benson's paper published in 1933: "A female past middle age had had severe asthma dating from an attack of lobar pneumonia in 1913. Sputum vaccine containing streptococcus and pneumococcus gave relief in this early phase of her infectious asthma. Her asthma recurred intermittently until 1925. At this time she consulted Dr. Alexander Fleming at St. Mary's Hospital in London. Dr. Fleming cultured the stool and found large numbers of streptococci of the viridans type and started vaccine treatment with small doses. Perfect relief occured in three weeks and lasted without incident over a period of five years. In 1930 she suffered another mild attack which responded promptly to a similar stool vaccine made by me. This case affords a striking example of the outstanding infectious nature of this group of asthmas."

Considering what bronchoscopic aspiration can and can not do for the asthmatic, we should first consider the pathology of asthma. Asthma may be due to bronchospasm, to mucosal edema with submucosal infiltration, or to thick gummy exudate blocking the bronchi. All three of these factors may be operative in any given patient, but one of the three factors may be predominant. In most cases clinical and bronchoscopic observation will enable us to determine which of these three factors is chiefly responsible for the dyspnea.

Patients in status asthmaticus are adrenalin-fast. These patients are adrenalin-fast chiefly because their obstruction is not due to bronchospasm. The obstruction in such cases is due primarily to mucosal edema plus thick, tenacious, adherent exudate. The latter can be

removed by bronchoscopic aspiration and thereby the dyspnea is relieved. We should clearly realize that bronchoscopic aspiration helps only by removing the gummy obstructing exudate. It may temporarily increase bronchospasm and it certainly does nothing to relieve mucosal edema and submucosal infiltration with inflammatory cells.

It is the patients who have persistent asthma for two weeks or longer who benefit by bronchoscopic aspiration. As a matter of fact, bronchoscopic aspiration during an attack of acute bronchitis or an acute exacerbation of chronic suppurative bronchitis with asthma can be hazardous because the bronchial mucosa is hyperirritable, and the presence of the bronchoscope is apt to produce additional severe acute bronchospasm with marked cyanosis. I had this happen several times until I learned better. For example, one patient was having only moderate dyspnea and no cyanosis. Even though cocainization was adequate, as soon as the bronchoscope was introduced the dyspnea became extremely severe and the patient became cyanotic. The cyanosis cleared up as soon as the bronchoscope was removed. In asthmatic patients having moderate dyspnea, it is always wise to administer aminophylline intravenously and epinephrine, 0.3 cc subcutaneously, a half hour before the bronchoscopy.

CONCLUSIONS

1. Asthma that begins after the age of 40 is usually due to infectious bronchitis. Adequate management of these patients having bacterial allergy includes elimination of all foci of infection.

2. High eosinophil counts are found in the blood and bronchial exudate of patients having infectious asthma.

3. Penicillin by intramuscular injection is effective treatment provided large doses are used and the organisms are moderately sensitive.

4. Cortisone is a useful adjunct for temporary relief.

5. Coexisting sinus infection usually requires effective surgical treatment.

6. Bronchoscopic aspiration is useful to remove gummy exudate and to obtain a reliable specimen for sensitivity cultures and autogenous vaccines.

7. Use of autogenous vaccines is an effective and much neglected method of treatment.

GEO. GEISINGER MEMORIAL HOSPITAL.

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DISCUSSIONS

Dr. Joseph P. Atkins (Philadelphia, Pa.): I sometimes think we expect too much of antibiotics such as expecting them to be effective in the case of inadequate drainage. It is my feeling that if the patient is constantly unable to clear his tracheobronchial tree of secretions, it is asking a good bit of an antibiotic to clear up that infection. Certainly, one would not expect an antibiotic to clear up an infection in a patient with a foreign body, so long as the foreign body remains there. I think of obstructing secretion in the tracheobronchial tree in very much the same manner as one would a foreign body.

If bronchoscopy, therefore, is looked upon not only as a diagnostic procedure, not only as a means of giving the patient mechanical relief to the airway, but, over and above those things, it should be understood that it gives the bronchi drainage. This permits whatever other therapy you are using, (whether it be antibiotics, vaccines or antispasmodic therapy) to be more effective. The fact that you are getting drainage allows the natural defenses of the bronchial mucosa to recover and become effective. For that reason it seems rational to me to carry out bronchoscopy in these patients where they have a tendency to retain viscid secretion, and to repeat bronchoscopy at relatively short intervals on several occasions, in order to maintain this factor of bronchial drainage.

Dr. Walter H. Maloney (Philadelphia, Pa.): I would like to take this opportunity to show a short film, a movie on bedside bronchoscopy which I feel

is particularly adapted to these patients with asthma, or severely dyspneic patients who cannot tolerate too much movement or manipulation, and in whom the problem of the position for bronchoscopy is a factor.

The film is made to show the pre- and post-operative use of the bronchoscope in cardiac patients, but I think it is particularly adapted to the asthmatic patient. I don't mean to intimate that this procedure is new. I merely assume that perhaps some of you have not used it.

(Showing of motion picture)

The patient is in bed, semi-elevated. This can be done in the patient's room or in the operating room, as you choose. The endoscopic instruments are carried on the mobile Jackson unit.

Local anesthesia is 4% cocaine. The patient has a pillow behind the shoulders. I am standing, either on the crossbars or on a bedside stool. Oxygen is insufflated through an oxygen channel. I feel that as little restraint as possible to these patients is important.

Dr. Herman J. Moersch (Rochester, Minn.): I am fully aware that there is a marked difference of opinion among investigators as to the cause of asthma. There are, of course, physicians who believe that all asthma is allergic in origin. I will not try to consider this factor, but would like to limit my remarks to infectious asthma which does not respond to the therapy that Dr. Davison has described. This group includes patients who invariably have had recurrent chills and fever. In many instances, temporary relief can be obtained by the administration of antibiotics, but the patients invariably will have further difficulty. On bronchoscopy, one not infrequently will find a definite stenosis of a bronchus. The bronchus most often involved, in my experience, is the superior division of the right or the left, lower-lobe bronchus. In opening a bronchus of this type, one invariably will obtain a small amount of retained secretion which is usually mucopurulent in character. Almost immediately after dilatation and aspiration, the patient will note an improvement in his condition.

Two of my associates recently carried out a study of 250 patients who had asthmatic bronchitis and had had recurrent chills and fever. At the time of bronchoscopic examination, bronchostenosis was found in 40 per cent of these patients. The thing that interested me most of all was that 68 per cent of these patients felt that their condition was definitely improved by the procedure. One should keep in mind that these were patients who previously had tried other forms of therapy. Frankly, I don't know just how to pick out the cases in which good results will be obtained, but, if a stenosis is found, I think there is an excellent chance of helping these unfortunate patients.

I also would like to emphasize a fact that Dr. Davison and Dr. Maloney pointed out, namely, that bronchoscopy is a hazardous procedure in a case of severe asthma.

Dr. Shirley H. Baron (San Francisco, Calif.): I just wanted to say that I enjoyed Dr. Davison's presentation very much, and I should like to ask him a question, as to just how he arrived at the large dose of penicillin—from 3 million to 5 million. Why not from 1 million to 2 million, or 4 million to 8 million?

Did you have some index for arriving at that particular dose in 24 hours?

Dr. Gabriel Tucker (Philadelphia, Pa.): I just wanted to mention that some patients in status asthmaticus have partial atelectasis of one or more lobes.

In the earlier days of bronchoscopy we had many of these cases. In this type of case you save the life of the patient because the dyspnea is due to atelectasis. I would like to recite one case.

This man was an eminent physician in the Philadelphia district. He had many troubles, including diabetes and the amputation of one leg, but he was still

quite active in his medical field. In consultation with his surgeon who had amputated the leg, and with one of our most eminent allergists in Philadelphia, Dr. Richard Kern, we finally decided that this man should be bronchoscoped.

He obtained so much relief when he would get these attacks of status asthmaticus that he called on us very frequently. He was given the then recognized proper treatment for asthma, but every once in a while he'd get blocked and call me, sometimes in the middle of the night. He would start for the hospital, and I would meet him there and bronchoscope him. Then he would be all right for a few days. He obtained immediate relief when large quantities of viscid secretion were aspirated. Being a physician, he knew pretty well what he was up against, and he didn't hesitate to come in and have himself cleaned out whenever this condition developed.

I don't believe status asthmaticus was mentioned, but in some of these patients there is atelectasis in the lower portion of the lung, and if you don't do something about it, mechanically, the patient is going to have serious difficulty.

DR. F. W. DAVISON (Danville, Pa.): Dr. Atkins, 30 of these 50 people did have bronchoscopic aspiration, some 6 to 10 times, so I do recognize its value in removing the type of exudate shown in the test tube in the first slide.

I am glad Dr. Moersch described the type of patient who had chills and fever due to inflammatory stenosis of one or more bronchi. I see relatively few who have stenosis limited to one bronchus. I more often see diffuse inflammatory edema. He is quite right, some of these patients have inflammatory stenosis, and benefit by dilatation and bronchoscopic aspiration.

In answer to Dr. Baron's question, I use sensitivity tests as an index of how much penicillin to give. It has been shown that if you give 5 million units a day, the average blood level is about 5 units. If I have patients whose organisms are sensitive to 5 units, I will give 5 million units as the minimum daily dose. For some organisms the bactericidal concentration is 20, 30 or 40 units per cc. Of course, when the resistance gets that high, it's rather futile to rely on penicillin.

I want to thank Dr. Tucker for mentioning the value of bronchoscopic aspiration for people who have patchy atelectasis. I didn't mention that specifically, but I did mention Loffler's syndrome, and these patients often have patchy atelectasis.

LXXVIII

REMOVAL OF RADIO OPAQUE FOREIGN BODIES FROM THE BRONCHI UTILIZING THE STEREOSCOPIC FLUOROSCOPE

ALDEN H. MILLER, M.D.

LOS ANGELES, CALIF.

The stereo-fluoroscope was first used in removing a radio-opaque foreign body from a bronchus in 1948.[1] This foreign body was a straight pin in the left lingular area which could not be visualized nor contacted with repeated ordinary bronchoscopic examination. Since that time three more straight pins, a very small piece of glass from a soda pop bottle, a tiny eyelet, a florist's corsage pin and a little screw have been removed from bronchi in patients at Children's Hospital in Los Angeles. Standard bronchoscopic procedures had not permitted these foreign bodies to be seen or contacted. The successful removal of these foreign bodies was possible only with the guidance of a fluoroscopist using a stereoscopic fluoroscope. This fluoroscope, affording three dimensional visualization of the foreign body and of the bronchoscopic instruments has been demonstrated on manikins to a number of endoscopists. Our experiences with this machine at Children's Hospital may be of interest in evaluating the merits of this new invention in comparison with other fluoroscopes as to ease and speed in removing foreign bodies, safety to the patient and safety of the operating personnel.

The stereo-fluoroscope is a fluoroscope that produces images having three dimensions. It uses two x-ray tubes, which operate on opposite halves of the cycle of the alternating current. These tubes are spaced so that their positions correspond respectively with the two different positions successively occupied by a single x-ray tube in making a stereoscopic pair of radiographs. The fluoroscopic images cast by the two tubes thus have the same relationship to each other as do the two radiographs in a stereoscopic pair. Although both images appear on the same screen, they are produced alternately instead of simultaneously.

In the usual use of the machine a special optical device causes the right eye to see the images cast by the left tube and the left eye

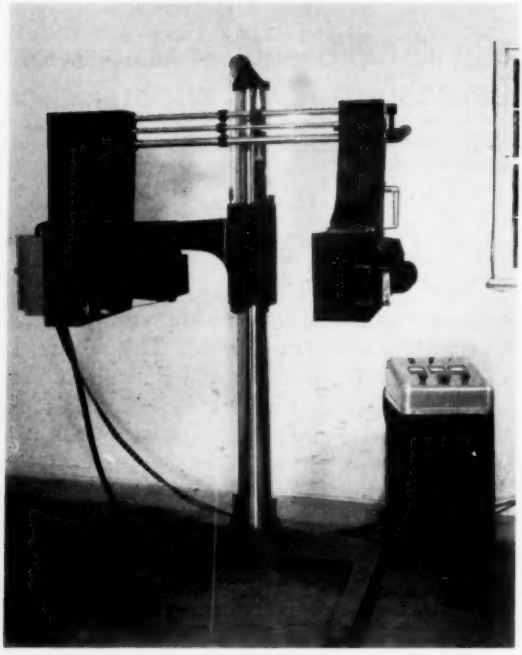

Fig. 1.—Stereo-fluoroscope oriented for vertical fluoroscopy.

to see the images formed by the right tube. When the radiologist's head is positioned at this eye piece, each eye sees through a different aperture and these open and close alternately and in synchronism with the operation of the x-ray tubes. The observer, however, is not conscious of this opening and closing, and each eye appears to be seeing continuously.

The instrument is arranged so that the adjustments to obtain stereoscopic images are made automatically. For instance, if the screen is moved away from the x-ray tubes, the tubes automatically move further apart so that their separation will correspond with the stereoscopic shift used in making radiographs.

Separate diaphragms are provided for the two x-ray tubes to control the spread of the beams, but the two sets of diaphragms are operated by a single set of controls. The beams can never get beyond the confines of the screen.

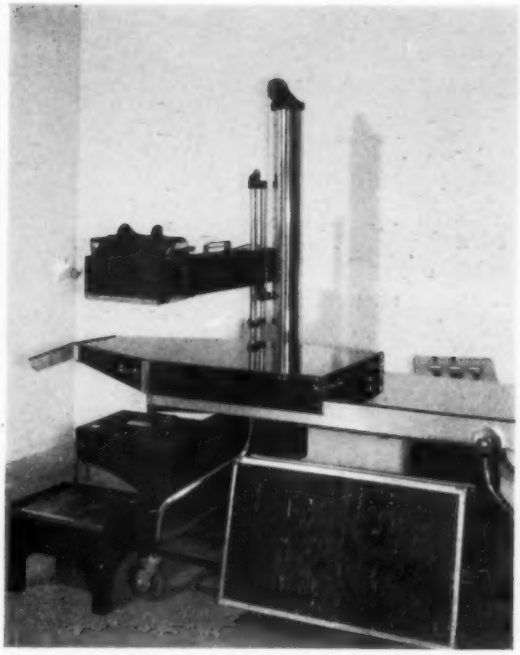

Fig. 2.—Stereo-fluoroscope positioned for horizontal fluoroscopy and endoscopy.

In order that the patient's skin may receive a minimum of radiation, the distance from the screen to the tube targets is much greater than the screen to target distance on the conventional fluoroscope. This distance on the stereofluoroscope is variable from 30 to 42 inches (76 to 107 cm).

The machine now uses 2 mm of aluminum as its standard filter. The radiation of our machine has been measured by a radiation physicist registered with the Radiological Society of North America and the United States Bureau of Standards. An identical machine was also measured by a physicist of a very large military hospital and the inventor of the machine has informed us that this physicist's measurements corroborated those of the physicist here.

The radiation received by the patient and the secondary radiation received by the operating personnel is substantially the same when

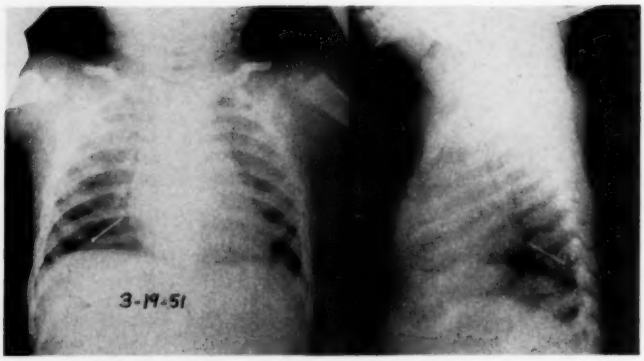

Fig. 3.—Straight pin removed with stereo-fluoroscopic guidance.

using the stereo-fluoroscope as in the use of a conventional instrument, despite the fact that two x-ray tubes are necessarily used in order to produce stereopsis. Each tube is on only half of the time and therefore the two tubes do not produce any more x-radiation than one tube on a fully rectified x-ray machine using both halves of the alternating current.

The control stand is equipped with a cumulative timer, which enables the radiologist to know how many roentgens have been received by the skin of the patient.

Extremely long screen-to-target distances are used with the stereofluoroscope in order to keep distortion in the image at a minimum. In order to do this ten millamperes of current are used in the operation of the stereofluoroscope. Usually 3 to 5 milleamperes are used with the ordinary, or biplane fluoroscope. However, the longer screen-to-target distances keeps the radiation at the patients skin at a low value and measurements of our machine reveal that the child patient receives only 7.1 roentgens per mintue to his skin with a 10 milleamperes setting and a 60 kilovolt peak and a screen-to-target distance of 30 inches. The average adult would receive 8.3 roentgens with a 65 kilovolt peak and a very thick adult 13 roentgens with a 75 kilovolt peak. In our seven successful procedures the patients respectively received 31 r, 8 r, 25 r, 12 r, 11.5 r, 62.2 r and 110 r.

The patient is the only one receiving any direct x-radiation. The endoscopist, the head holder and others are subject to the same

Fig. 4.—Small screw removed with stereo-fluoroscopic guidance.

secondary irradiation present in any fluoroscopy. The fluoroscopist wears a lead rubber apron and lead rubber gloves; the endoscopist wears a lead rubber apron. A special stool protects the legs of the fluoroscopist from any secondary irradiation that may come from the under side of the table.

The following measured values of secondary radiation show that the stereofluoroscope compares favorably with any other fluoroscope in the matter of safety to personnel.

The radiation at the observer's eyes at 65 kilovolt and 10 milleamperes, using the 30" screen-to-target distance and a phantom 20 centimeters thick with the shutters or diaphragm all the way open, is .008 roentgen per hour—certainly a negligible amount.

The radiation at the observer's shoes, without using the shield provided by the special stool, at 70 kilovolt and 10 milleamperes, is

Fig. 5.—Tiny rivet, behind heart shadow removed with stereo-fluoroscopic guidance.

.001 roentgen per hour. In other words, it would take 1000 hours for the observer's shoes to receive one roentgen.

The endoscopist himself is usually in a position where the secondary radiation is the greatest. With the shutters all the way open, the secondary radiation at 1 foot in open air from the portion of the patient that is receiving the direct rays is .072 roentgen per hour. This means that it would take fourteen hours of actual radiation time for the endoscopist to receive one roentgen of secondary radiation. Actually, the x-rays are usually on only about one-third of the time that the endoscopy is in process. This indicates that less than one roentgen would be received by the endoscopist during 42 hours of endoscopy. It is considered perfectly safe for those using x-rays constantly to receive one roentgen per month. It would consequently be possible for an endoscopist to use the stereo-fluoroscope for approximately 1½ hours every day without obtaining an amount of radiation that would be considered objectionable by the most exacting standards.

A total of thirteen procedures with stereo-fluoroscopic guidance has been performed on eight patients at Children's Hospital. Two of these patients were adults. The removal of the foreign bodies was successful in all patients with the exception of one straight pin which had worked itself into the peripheral lung parenchyma of the right lower lobe so that it was "out-denting" the diaphragmatic surface when the foreign body was removed by thoracotomy. There

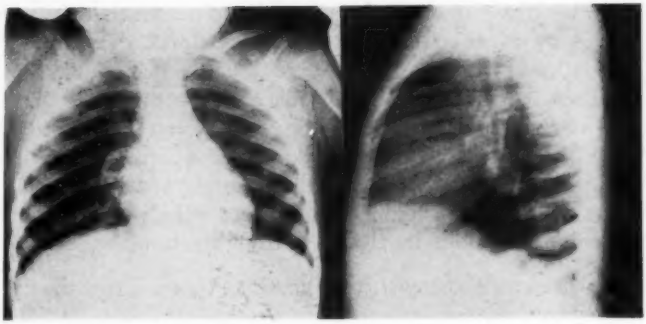

Fig. 6.—Bit of glass bottle removed with stereo-fluoroscopic guidance.

was only one complication in the thirteen procedures performed on these eight patients. In this one patient a subcutaneous emphysema and a mild emphysema of the mediastinum developed following the removal of a straight pin from the right lower lobe. There was complete spontaneous recovery in four days. Only one patient developed an erythema of the skin over the anterior chest—this was a patient who had received 300 r in an unsuccessful attempt to remove a straight pin. Five foreign bodies were removed with one attempt; two necessitated a second procedure.

The seven successful removals were made in 30 seconds, 50 seconds, 1 minute, 2 minutes, 2½ minutes, 4½ minutes and 28 minutes following the beginning of the fluoroscopic guidance. In the unsuccessful atempts the branchoscope was never down longer than 30 minutes. These figures attest to the ease and speed that this stereo-fluoroscope has afforded us in removing these foreign bodies. The three dimensional visualization of the foreign bodies and the bronchoscopic instruments allowed the fluoroscopist to at once determine whether or not the forceps were in the correct bronchus in the plane of the foreign body. Much time was saved by not having to switch the fluoroscopic screen from one position to another and back again numerous times. It seemed to us this was a decided advantage when compared to our previous experiences with a biplane fluoroscope in cutting down the length of time that the bronchoscope had to be in place as well as in reducing the total exposure of all concerned to irradiation.

A number of interesting observations and deductions have been made from our experiences with the above foreign bodies: The fluoroscopist must have stereoscopic vision himself; much experience in viewing x-ray films stereoptically is very helpful. Very good dark adaptation must be maintained at all times once the fluoroscopic visualization has been started. While the fluoroscope is turned on there is no light in the room whatsoever. While the fluoroscope is turned off and the endoscopist is changing positions, forceps, aspirating or viewing the bronchi only the ordinary red "safety" light is on. The fluoroscopist should keep the foot switch going off and on. The machine is on no more than a third of the time.

Another bronchoscopist can sometimes better direct the operator as his instructions are interpreted easier.

The fluoroscopist can immediately tell the endoscopist when his forceps have entered the bronchus in which the foreign body is lodged. The foreign body moves widely about with respirations in a fan shaped area until the forceps enter the correct bronchus. Then the foreign body becomes stationary.

When the patient is lying on the table on his back the foreign body sometimes is over his vertebrae in which case ordinary fluoroscopy, or the vertical beam of the biplane fluoroscope, would not permit it to be visualized well. Only with the stereo-fluoroscope can this be overcome by changing the fluoroscope to a side to side horizontal position without moving the patient.

Fortunately for us the stereo-fluoroscope has given an excellent image of substances sometimes difficult to visualize with any fluoroscope. Two tiny bits of glass and a splinter of chicken bone have been easily localized and removed.

The stereo-fluoroscope seems to us to be most useful in procedures other than just the removal of foreign bodies from the bronchi. A tiny piece of glass has been removed from the heel of a boy and broken off needle points have been removed from the foot and the back and a chicken bone splinter from the foot—all in patients upon whom unsuccessful surgical removal had previously been attemped.

The inventor of the machine has personal communication from the staff of a tuberculosis sanitorium where the only other stereo-fluoroscope is in clinical use. There, stereo-fluoroscopy has been more than ordinarily helpful in the localization of adhesions between the visceral and parietal pleura, in the localization of residual empyema cavities and lipiodol studies.

CONCLUSIONS

Stereo-fluoroscopic guidance has made possible safe, easy removal of radio-opaque foreign bodies from the bronchi when visualization or contact was impossible with ordinary bronchoscopy. It is suggested that three-dimensional fluoroscopy can be of great aid in many other fields.

The technical description of the stereo-fluoroscope is by its inventor, Mr. Leroy Leishman, of Los Angeles.

From the School of Medicine, University of Southern California and Children's Hospital Society of Los Angeles.

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DISCUSSIONS

Dr. Murdock Equen (Atlanta): Great progress has been made since I read a paper on the biplane fluoroscope at our Charleston meeting in 1935, this progress culminating in the three dimensional fluoroscope that Dr. Miller has just demonstrated.

I note that he did not mention the magnet. I would like to put in a plug for this very useful litle instrument.

Last night Dr. Dixon invited me to discuss this paper and I called Atlanta to ask for some slides. Unfortunately, the slides received illustrate only intragastric and intraduodenal cases, but since they show also the use of the fluoroscope, I hope Dr. Miller will let me present them.

(Slide) First, we will show the strength of this magnet. It is a product of General Electric Company and the models used by me were finished by Georgia Tech according to my designs. The magnet will lift twenty-five times its own weight.

Dr. Equen then showed slides of a table and a magnet.

For intrabronchial work we use a curved magnet fixed on a semirigid tube so that it can be turned into either the right or left main stem bronchus. It can also be used in the stomach, for under fluoroscopic guidance the curved magnet will follow the curvatures of the stomach easily. In small children a straight magnet of smaller diameter, attached to a ureteral catheter, can be passed through a 3 mm bronchoscope and in adults, such a magnet can be introduced under fluoroscopic guidance into the smaller bronchi to pick up magnetic foreign bodies that one cannot see by direct vision. It has largely replaced the use of forceps in the removal of magnetic foreign bodies from the tracheobronchial tree in our Clinic.

(Slide) For intragastric or intraduodenal work we also use a curved magnet, attached to a radiopaque string, so as to show up under the fluoroscope. A child can swallow such amagnet for the removal of foreign bodies from the stomach or duodenum and one can see just exactly how much string to give him, by following the progress of the magnet. If you give him too much string, it is likely to get tangled up. We immobilize the child's arms because if you do not he will pull the magnet out. You give him enough string to make contact with the ingested foreign body, and when the fluoroscope shows contact between the magnet and the foreign body, both are withdrawn.

I would like to ask Dr. Miller to give an approximate idea of the cost of the machine. I am afraid that for the average bronchoscopist the expense may prove prohibitive.

Dr. Edwin N. Broyles (Baltimore, Md.): It has been my pleasure to have been in Los Angeles and have had this machine demonstrated to me by Dr. Miller. We didn't have a patient that day, but we certainly got a wonderful stereoscopic view.

There is only one thing about which I would like to ask Dr. Miller. As the inventor stated, I believe, the phase of the machine may change, so that instead of moving to the right, you apparently move to the left. I guess there is some way to check yourself to see just exactly how that phase is.

Will you explain that, Dr. Miller, please?

Dr. DeGraaf Woodman (New York, N. Y.): I don't want to throw cold water on this, because I saw this machine and it does all that Dr. Miller says it does. I was so enthusiastic about what I saw that we ordered one for the Columbia-Presbyterian Medical Center. We had it for a few months. There are three of us who do the Bronchoscopic work and we took turns practising on dogs with foreign bodies. The visibility was not satisfactory. We could not see through the heart shadow. Its action was not constant.

The local company in New York who undertook the management and servicing of the machine didn't seem to be able to make it work, so we called the inventor who came from the West coast. He came and found one or two things wrong and replaced some parts. It worked a little better but we still weren't happy about it.

Finally thinking that perhaps we were at fault we called on Louis Clerf and his x-ray man Dr. Swenson. They came up from Philadelphia to verify our findings. Clerf took a look at it and said "I just as soon look through a Brownie Camera."

I saw this work well in Los Angeles and therefore am convinced that we had a defective machine. I would like to warn that inasmuch as this is a big capital outlay, anyone planning to take this on should do so through a reliable local company who can adequately service it and I would also suggest that they not buy one without taking it on a trial basis.

Dr. Albert H. Andrews, Jr. (Chicago, Ill.): I had the opportunity of discussion this subject with Dr. Miller last night, and I was very much impressed by what he told me.

The thing that disturbed me most was my experience with stereo films, and the recognition of my inability to judge depth accurately in the steo film. Upon meditating overnight on this, I think that perhaps I have worked it out.

There is a difference between stereo vision and depth perception, and from the endoscopic standpoint it is depth perception which is of paramount importance. Stereo vision is, of course, one element in that, but probably because this is fluoroscopic, and because there are the other relationships between the ribs, heart and vertebral column that are apparent to the fluoroscope screen—the slightly imperceptible movements of the head that are made in depth perception make this possible.

I think we ought to reserve a final opinion on this equipment until each of us has an opportunity to evaluate it, because there is considerable variation between us in our depth perception, and perhaps in our ability to use an instrument of this type. I think we must recognize that it has real advantages when compared with the biplane fluoroscope.

Dr. V. K. Hart (Charlotte, N. C.): This is certainly a beautiful presentation, and it gives one an inferiority complex when he has to work with some of the old equipment.

I would like to mention this, though, that in our small hospital, for over 15 years, we have used the principle—and very satisfactorily—of the single screen. We have done this simply by mounting two x-ray tubes beneath the ordinary fluoroscopic table. You then get two images on the screen, viz., two foreign bodies and two pairs of forceps. By the principle of the divergence of rays, the fluoroscopist can tell one at once whether he is above or below the foreign body, or right or left, and as soon as you enter the correct bronchus, the images tend to coincide.

We have found this a very satisfactory method for the average foreign body beyond bronchoscopic vision. However, I doubt very much whether we could have handled that small piece of glass by this method. We might have.

My chief reason for mentioning this is for the benefit of those of us who cannot afford very large capital outlays. Here is a system which does not involve such terrific expense.

DR. ALDEN H. MILLER (Los Angeles, Calif.): I want to first thank all of my discussants.

Yes, Dr. Equen, we did use your magnet—a small, Alnico magnet—on the first two straight pins, but we couldn't get the magnet within 2 to 3 centimeters of the head of the pin. In fact, the second straight pin was out in such a tiny bronchus that we had an engineer make for us a straight grasping forceps, the jaws of which were only 3 millimeters in their greatest diameter. So you can see the pin was in such a small bronchus that we couldn't approach it with the magnet.

Something new has been added, Dr. Broyles, since you were there.

You must mean "reversal of the image." I don't know exactly what that means, except that in the case of this fluoroscope, when you first turn it on, it makes a difference which half of the cycle is used first, and so there was a 50-50 chance that your image would be reversed anterior-posteriorly.

That very thing happened in one of our unsuccessful attempts. Dr. Karsner, our fluoroscopist and radiologist, just couldn't understand why I couldn't follow his directions. Pretty soon the 250 r was used up. Then the inventor put a nickel on the child's chest, took a look, and said, "We forgot to look for and correct a reversal of the image."

Dr. Andrews: I think that just as long as the fluoroscopist has good stereoscopic vision, he doesn't have to worry about depth perception as it is almost entirely dependent on stereoscopic vision. All you have to do it to concern yourself with the relationship of the ends of your forceps and the end of the pin. The fluoroscopist gets such a beautiful 3-dimensional vision of both that I don't think he is conscious of the depth.

Dr. Hart: I can't comment, except to say that your method seems very ingenious. I have had no experience with it at all.

Somebody wanted to know about costs. I can just guess at it, in round numbers. I would say around $10,000.

Finally, Dr. Woodman. I am very happy that Dr. Woodman made his comments, just as I am happy to have had this opportunity to talk to you about this instrument because I have been so enthusiastic about it. I'm sorry that my enthusiasm rubbed off on Dr. Woodman. When I got letters from him about their poor results, I felt very badly.

That is the main thing I want to stress—I am not advising a single one of you to acquire this machine. It's just that our experiences have been so wonderful with it that I wish you would think about it when you do think about this type of equipment.

My own personal guess is exactly the same as Dr. Woodman's, that there was something wrong with that individual machine.

I showed his letter to Dr. Karsner, our roentgenologist, and he could not understand it at all, because, believe me, it has been the simplest and easiest fluoroscope for us to use.

LXXIX

AMYLOID TUMORS OF THE LOWER PART OF THE RESPIRATORY TRACT AND MEDIASTINUM

HERBERT W. SCHMIDT, M.D.*

JOHN R. MCDONALD, M.D.**

AND

O. THERON CLAGETT, M.D.***

ROCHESTER, MINN.

Amyloid disease was described more than a hundred years ago by Rokitansky[1] and by other authors. They described a condition in which various organs of the body were infiltrated with a firm, waxlike, homogeneous material. This condition in the past was called "lardaceous disease" or "bacony or waxy degeneration."[2] It was found that the material that infiltrated the various organs involved in this disease would become blue-black when treated first with iodine and then with dilute sulfuric acid. Because of its resemblance to starch it was suggested that this material be called "amyloid."

In the past, it was felt that amyloid disease was a degenerative process. However, in some instances at least, it appears that it is an infiltrative rather than a degenerative process.[3] No one knows how amyloid originates or how it is deposited.

Various classifications of amyloid disease have been given, but in general one can speak of two types as indicated in the following outline:

1. Primary amyloidosis
   a. Systemic
   b. Tumor-forming
2. Secondary amyloidosis
   a. Infections: tuberculosis, empyema, bronchiectasis, pulmonary abscess, actinomycosis, osteomyelitis, tabes dorsalis, chronic ulcerative colitis, dysentery, regional

*Division of Medicine. **Division of Surgical Pathology. ***Division of Surgery, Mayo Clinic.

enteritis, subacute bacterial endocarditis, pyelonephritis, pemphigus, malaria, leprosy, rheumatic fever

b. Tumors: multiple myeloma, carcinoma of bronchus, carcinoma of stomach, Hodgkin's disease, leukemia

c. Other causes: rheumatoid arthritis, diabetes mellitus, thermal burns, cirrhosis of the liver.

It is beyond the scope of this paper to discuss secondary amyloid disease.

Cases of primary systemic amyloidosis were presented by Königstein[4] in 1925 and by Lubarsch[5] in 1929. The condition is rare—approximately 70 cases had been reported by 1950.[6] In the great majority of these cases the diagnosis was not made until postmortem examination. This type of the disease has the following characteristics: 1. There is no pre-existing disease which might have caused it. 2. Amyloid is deposited chiefly in tissues of mesodermal origin such as the cutis, heart muscle, striated muscle of the tongue, skeletal muscle, smooth muscle of the gastrointestinal tract, urinary bladder, and arterioles.[7] (In secondary amyloid disease, amyloid is deposited chiefly in the spleen, liver, kidneys and adrenals.) 3. There are atypical reactions to some of the specific histologic stains for amyloid.

Amyloid deposits may produce skin papules, heart failure, macroglossia, muscle pains or gastrointestinal symptoms of various types depending on the part of the gastrointestinal tract that is affected. Patients who have primary systemic amyloid disease are older than patients who have secondary amyloidosis. Death occurs in the sixth decade of life most frequently, and the sexes are affected equally. Death in approximately one half of cases is due to involvement of the heart. The results of the intravenous Congo red test may be negative in this type of amyloid disease.[6]

The Congo red test is based on the fact that certain types of amyloid deposits are capable of removing measurable amounts of the dye, Congo red, from the blood. The test as originated by Bennhold has been modified in various ways. All similar tests that have been used clinically in the diagnosis of amyloid disease have two weaknesses: 1. Some Congo red is removed from the blood by nonamyloid substances. This probably occurs in the reticulo-endothelial system. Such removal is appreciable, it is larger in some cases than in others, and it may cause false positive reactions. 2. Negative results may be obtained with the Congo red test even in cases of proved amyloid disease.

The Paunz test is a Congo red test in which a small amount of the dye is injected intravenously (1.2 mg per kilogram of body weight). The results are read "negative" or "positive" on the basis of whether the dye is present or absent in a blood specimen drawn one hour after injection of the material. There have been no appreciable number of false positive reactions with this test;[8] there are, no doubt, an appreciable number of false negative reactions.

Tumor-forming amyloid disease is a rare type of primary amyloidosis which occurs in patients who have none of the diseases that lead to secondary amyloidosis. Localized infiltration may occur in the heart, skin, larynx, trachea, mediastinum and bronchi. These amyloid tumors occur most frequently in organs that are made up in part of cartilage or that are subject to the frequent occurrence of abnormal calcification or ossification. This observation and the fact that calcification and osteoid tissue occur so often in amyloid tumors suggest that some common factor is present to cause deposition of amyloid, calcium and osteoid tissue. It has been suggested that chronic inflammation may be a causative factor. In tumor-forming amyloid disease, men are affected between two and three times as often as women.[9]

Because of the rarity of tumor-forming amyloid disease in the lower respiratory passageways and in the mediastinum, we are describing 3 cases in which the diagnosis has been made clinically at the Mayo Clinic. Cases 1 and 2 have been reported previously,[10, 11] but we should like to introduce them into this discussion of the subject and give a progress note on them.

REPORT OF CASES

CASE 1.—A white man, 35 years of age, a welder came to the Mayo Clinic on January 10, 1946. He had a cough of nine years' duration, and had been coughing up blood for one week prior to admission. His past history revealed that in 1937 he had experienced pleuritic pain over the right side of the thorax, without fever. A morning cough productive of purulent, occasionally blood-streaked, sputum had developed. Shortly after the onset of this illness, bronchography had been performed and a diagnosis of bronchiectasis of the right lung had been made. He had experienced recurrent chills and fever and pleuritic pain. In 1942, he had had one episode in which he had coughed up approximately a cupful of blood. A week prior to admission to the clinic, he had begun to cough up blood each day.

Fig. 1.—Area of infiltration in the right lower and middle lobes.

Stereoroentgenograms revealed a diffuse infiltration of the lower and middle lobes of the right lung (Fig. 1). The sputum was negative for acid-fast bacilli. Bronchoscopic examination disclosed a large blood clot in the right lower lobe bronchus. Bronchograms revealed unsatisfactory filling of portions of the right middle and right lower lobe bronchi. It was thought that the patient probably had bronchiectasis of the right lower lobe, and that the right lung should be explored. On exploration there was extensive induration and distortion at the hilus, and a mass was found. On palpation, this felt too hard for carcinoma, but still it did not simulate an enlarged hilar lymph node. It was thought advisable to perform pneumonectomy, and this was done. After operation, urinalyses were repeatedly negative for Bence Jones protein. The Paunz test for amyloid similarly gave negative results. The concentration of serum protein was 6.4 gm per 100 cc. The albumin-globulin ratio was 1.6:1. Two months after operation, the patient had no symptoms and had resumed full activity.

In the specimen removed at operation there was a hard, irregular, infiltrating, white-yellow mass about the bronchi, the right pulmonary artery, and the inferior pulmonary vein. The infiltrating tumor extended laterally and inferiorly into the substance of the

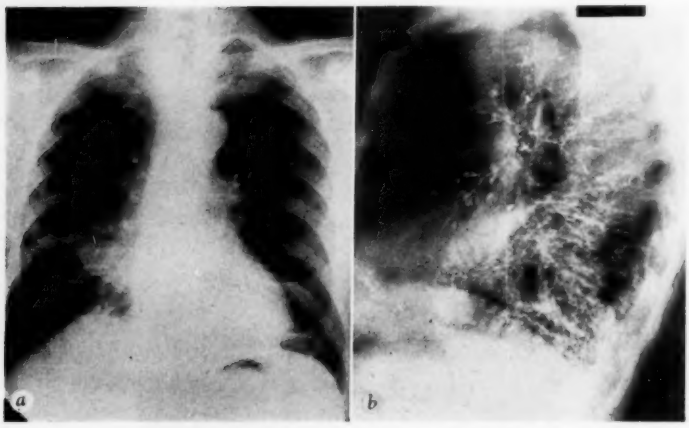

Fig. 2.—Tumor of the right middle lobe. *a*. There is complete collapse of the right middle lobe. *b*. Lateral bronchographic studies of the right lung show obstruction to the middle lobe bronchus at a point 2 cm distal to the site of origin.

lung. It extended out around the bronchus to the middle lobe for about 3 cm and out around the right lower lobe for about 5 cm. The mass was very hard to cut, having the consistency of leather, and small calcified flecks were seen in the mass. The infiltration involved the walls of the right middle and lower lobes. Microscopically, the mass was found to consist of amyloid, and there was calcification of the homogeneous material throughout. Lymph nodes taken from the hilar region did not contain amyloid. A letter written March 1, 1953, informed us that the patient was feeling well and was working every day.

Case 2.—A retired telephone lineman, 66 years of age, came to the Mayo Clinic on November 21, 1946, because of a tumor of the right lung. He had experienced a mild cerebrovascular accident three years previously but had made an uneventful recovery from this. Seven weeks before admission he had noticed the development of midepigastric burning during exertion. This had increased in severity until, at the time of admission, it could be precipitated by walking one block. A mildly productive cough had been noticed six weeks prior to admission. The sputum had never been bloody. A tumor was seen in roentgenograms of the thorax and the patient was referred for the consideration of surgical treatment (Fig. 2*a*).

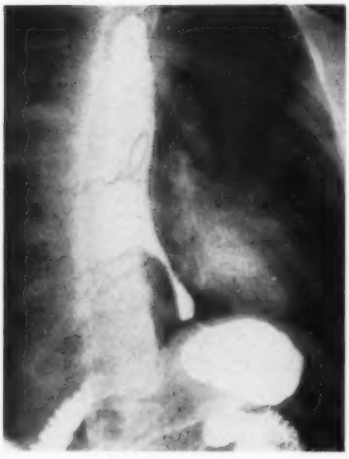

Fig. 3.—Extrinsic pressure is being exerted on the distal part of the esophagus.

On examination, the thyroid gland was enlarged and a fine tremor of his hands was noticed. The basal metabolic rate was +30 per cent. After the administration of Lugol's solution the basal metabolic rate was +25 per cent. On bronchoscopic examination no evidence of a tumor could be seen in that part of the bronchial tree that could be visualized. Bronchial secretions were studied for malignant cells with negative results. Bronchography revealed a filling defect of the right middle lobe bronchus (Fig. 2*b*). The Paunz test gave negative results.

It was thought advisable to do a thyroidectomy first, because of the evidence of hyperthyroidism. Subsequent to thyroidectomy, the basal metabolic rate was −10 per cent.

At exploratory thoracotomy a very hard mass was felt in the middle lobe of the right lung and it was noted that this lobe was airless. The right lung was removed. Pathologic examination revealed the mass to be stony hard. It was 6 cm in diameter, gray and black in color, and firmly embedded in the surrounding pulmonary tissue. The bronchus of the right middle lobe passed directly into the mass and was completely occluded by it. Microscopic examination indicated that the tumor consisted of amyloid.

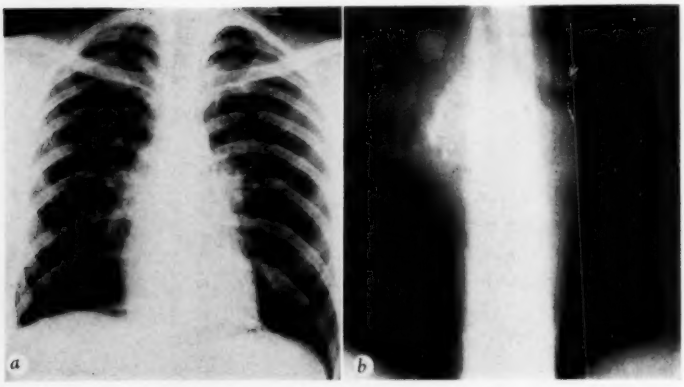

Fig. 4.—*a*. Routine roentgenogram reveals infiltration in the left upper lobe. There is widening of the mediastinum due to the tumor. *b*. Tomogram reveals a partially calcified mass in the mediastinum which is causing obstruction to the right main and intermediate bronchi and to the left main bronchus.

The patient returned eleven months after the right pneumonectomy, complaining that his stomach filled up after taking small amounts of food. He also complained of regurgitation, and said that occasionally food would stick in the lower part of the esophagus. Roentgenograms showed narrowing of the lowermost 3 or 4 inches of the esophagus with compression on the right esophageal wall (Fig. 3). On esophagoscopic examination extrinsic pressure on the distal 3½ inches of the esophagus was seen. It was impossible to pass the esophagoscope into the stomach because of the extra-esophageal pressure. There was no break in the esophageal mucous membrane. Subsequently, the esophagus was dilated to the diameter of a 45 F. sound with very little resistance felt in the lower part. It was thought possible that amyloid deposits could be compressing the distal part of the esophagus. Roentgen therapy was given to the involved region, and the patient did very well following this treatment. He gained 29 pounds and lost his dysphagia and his distress after eating.

In a letter dated February 12, 1953, the patient stated that in general he was feeling well. He said that he had had an elevation of blood pressure in December, 1952, with a systolic reading of 220 mm of mercury, but that this had reached a normal range within

two weeks. He described a transient diplopia which occurred following this episode, but had given him very little trouble. At the time this letter was written the patient was 73 years of age.

CASE 3.—A white man, 34 years of age, came to the Mayo Clinic January 6, 1947. He said that while on duty as an army officer he had had what was diagnosed as lobar pneumonia in March, 1942. This had occurred about ten days after an inoculation to prevent yellow fever. He was hospitalized for twenty-eight days, after which he apparently recovered completely. A year and a half later, in the fall of 1943, he coughed up blood on several occasions. He was hospitalized, a bronchoscopic examination was performed, and a diagnosis of "tumor at the bifurcation of the trachea" was made. He was transferred to another Army hospital, where another bronchoscopic examination was made. The final diagnosis at that hospital was, "Lymphadenitis, mediastinal lymph nodes, probably calcified hilar nodes, chronic, cause undetermined, improved." Since the onset of hemoptysis in 1943, this patient had coughed up blood recurrently, varying in amount from a few drops to 2 tablespoonfuls. Five months prior to admission, pain that was aggravated by deep breathing had developed to the left of the sternum. He had lost 15 pounds in one year.

Roentgenologic examination was reported as showing linear fibrosis of the upper lobe of the left lung (Fig. 4*a*). Examination of the sputum for acid-fast bacilli gave negative results. Bronchoscopy revealed that both main bronchi, as well as the right intermediate and right lower lobe bronchi, were infiltrated with tumor. The left main bronchus was reduced to two-thirds of its normal diameter, and the right main bronchus was reduced to one-third to one-half of its normal diameter. There was marked widening of the coryna. The right main bronchus was irregular and infiltrated. Bronchoscopic biopsy of the right bronchial tree revealed amyloid disease (Fig. 5*a* and *b*). Tomographic studies following bronchoscopy disclosed a large mass, which contained calcium, below the coryna (Fig. 4*b*). Both main bronchi were constricted. Roentgen therapy was given over the mediastinum to see if it would have any effect on the tumor.

After his return home, the patient did not do well. He began to cough up from 2 to 3 ounces of foul, purulent secretion daily. His temperature ranged between 99° and 100° F., he had pleuritic pain, and he lost 10 pounds. When he returned to the clinic in March, 1947, roentgenograms of his thorax disclosed the presence

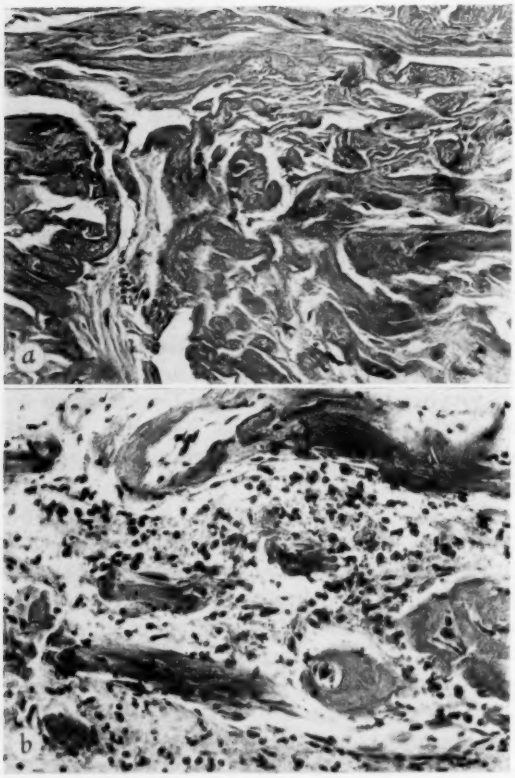

Fig. 5—*a*. Amyloid disease of the bronchus. Note the dense masses of eosinophilic amyloid (hematoxylin and eosin; x275). *b*. Note the amyloid disease localized to the walls of arterioles and the adjacent inflammatory reaction (hematoxylin and eosin; x275).

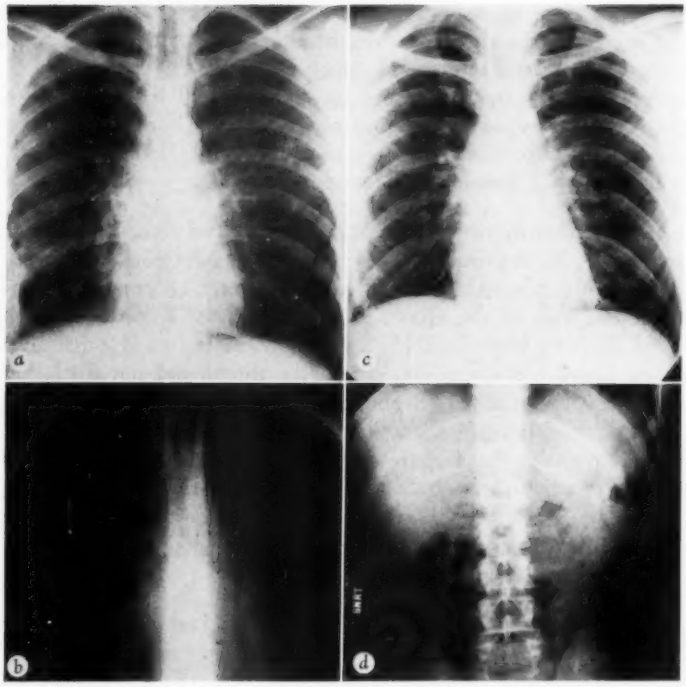

Fig. 6—*a*. Localized pleural fluid in the right axillary line. *b*. Tomogram reveals air in the mediastinal tumor. *c*. Roentgenogram taken five years and eight months after the first examination. *d*. Well-circumscribed mass in the superior portion of the right lobe of the liver.

of fluid in the right axillary line (Fig. 6*a*). Twenty cubic centimeters of thin, turbid, amber fluid were removed from the pleural space, and culture of this fluid gave negative results. The patient was treated with penicillin. On March 7, 1947, the results of bronchoscopic examination were much the same as at the time of the first bronchoscopy. Part of the tumor was excised, with biopsy forceps, from the right intermediate bronchus in an attempt to establish a better airway. Tomograms taken six days later revealed that air was present in the mass (Fig. 6*b*). It was assumed that the last bronchoscopic examination, with removal of tumor tissue, had created a bronchial perforation and a fistula to the tumor.

The patient did surprisingly well clinically. It was thought that if resection were to be performed it would mean that the right lung might have to be removed and that a plastic tube might have to be inserted into the left main bronchus to maintain an airway. We were all reluctant to advise an operation of this magnitude. On March 14, 1947, another bronchoscopy disclosed definite improvement. There was less evidence of obstruction in both main bronchi, and some decompression of the tumor apparently had occurred. It was decided to follow a conservative program.

The patient improved. At times he noticed some dyspnea with exertion but by September, 1948, he was able to work full time on his farm. By September, 1950, he had gained 25 pounds. On his most recent visit to the clinic, in September, 1952, he felt well but noticed dyspnea after climbing two flights of stairs. He had a dry, hacking cough. Roentgenograms of the thorax did not disclose any abnormality (Fig. 6*c*). Bronchoscopy revealed the main bronchi to be more widely open than they had been at the time of any previous examination, and there was some residual narrowing of the right intermediate bronchus. A nodular lesion, seen in the depths of the right lower lobe bronchus, was removed with biopsy forceps. This lesion was the site of an inflammatory reaction, with granulation tissue being formed.

It is of interest that the Paunz test gave negative results in this case. It is also of interest that roentgenograms taken of the lumbar region of the spinal column on November 15, 1948, showed a well-circumscribed mass in the liver (Fig. 6*d*). From a clinical standpoint it was impossible to know the nature of this lesion without surgical exploration of the liver. We were reluctant to do this. Subsequent roentgenograms have shown no change in the size of this mass. One of the possibilities that was thought of was that of another amyloid tumor.

All three of the foregoing patients have done very well clinically over a five-year period. Two of the patients received roentgen treatment to the region of the tumor, and one did not. Whether or not roentgen therapy had any beneficial effect is questionable. It appears that this type of amyloid disease runs a benign course unless the tumor infiltrates and causes bronchial obstruction. If this occurs, an attempt must be made to relieve the obstruction either by resection or by an attempt to improve the airway by removal of a portion of the tumor through the bronchoscope. In general, any therapy that is directed toward the relief of symptoms should be as conservative as possible.

SUMMARY

Three cases of amyloid tumor of the lower part of the respiratory tract and mediastinum have been presented. Two of the patients underwent pneumonectomy for this condition, since bronchogenic carcinoma could not be entirely excluded at the time of operation. The third patient was treated by bronchoscopic removal of part of the tumor. All 3 patients have done very well for more than five years following treatment.

MAYO CLINIC.

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DISCUSSIONS

DR. EDWIN N. BROYLES (Baltimore, Md.): Dr. Schmidt is to be congratulated on the report of these cases, especially the third case. I might say it has been my misfortune to have had a similar case, which I am sorry to say terminated fatally, and not with such good results as Dr. Schmidt has shown.

This was a young woman about 35 years old who had very marked dyspnea. Upon x-ray she did have a slight thickening in one lobe of her right lung, lower lobe, and upon bronchoscopic examination, the carina and the left bronchus bled very freely.

As far as I could tell, there was no definite tumor mass in the bronchial lumen, but it gave the impression that the bronchi were being constricted from something without.

I thought this girl ought to have an exploratory thoracotomy. That was refused. A year later she came back again to the hospital, this time definitely worse, with the bronchoscopic picture practically the same.

Dr. Blalock opened her chest this time, and found this iron-like mass that surrounded the carina and both primary bronchi. He could not resect it. He closed up, and just said, "I can't do anything about it." She died about four days following operation.

At autopsy the typical condition mentioned by Dr. Schmidt was found, with this extremely hard mass. I don't believe we found any amyloid in our case. There was just an extremely dense, hard scar tissue, with no definite ulceration. It must have pressed on some of the blood vessels that went to the left bronchus, because she bled freely from that area.

Perhaps we did the wrong thing in operating on her, although I do think it is wise in many of these cases, and especially in these undiagnosed ones. At least we have to have the chest surgeon do an exploratory thoracotomy.

DR. JOSEPH P. ATKINS (Philadelphia, Pa.): There is one question I would like to ask, and that is whether there is any evidence in these cases of primary systemic amyloidosis as well as the tumor-forming type.

We have seen one case in which the picture was that of primary amyloidosis, the patient's chief complaint being cardiac difficulties associated with the myocardial involvement.

The diagnosis would have been made by a biopsy of a nodule on the larynx. This particular nodule looked just like an ordinary vocal nodule. The patient was sent to us because of hoarseness. We declined to do anything about this nodule because of the very serious cardiac situation.

The diagnosis did not become completely apparent until the necropsy, at which time this small tumor on the cord was found to be completely infiltrated with amyloid, and would have given the diagnosis ante mortem.

DR. PAUL H. HOLINGER (Chicago, Ill.): We have had two patients with amyloidosis of the trachea and bronchi, and therapeutically these were largely endoscopic problems. The removal of the obstructing material endoscopically from the trachea or the bronchus is fortunately a relatively simple procedure. Since the material is butterlike, it allows for easy, repeated removal.

If there were systemic means of approaching the problem, I believe we would be more fortunate. However, I would like to question the indications for exploration and resection when there is the obvious amyloidosis within the lumen itself above the point of resection.

DR. WILLIAM V. LEARY (Houston, Tex.): As far as treatment is concerned, it has been recently suggested in an editorial in the Annals of Internal Medicine on primary systemic amyloidosis that another form of treatment might be helpful. Reference has been made to several cases of secondary amyloidosis which have apparently been improved or cured by the oral administration of powdered liver.

I can't say that the adminis ration of powdered liver appeals to me in general, very much, for any kind of disease, but it is suggested by Dr. Morgan, who wrote the editorial, that since we have no other form of treatment, possibly this should be done.

I realize that there is a difference between secondary and primary amyloidosis, but Dr. Morgan was talking about primary systemic amyloidosis.

The disease does seem to enjoy this distinction, that nobody has tried ACTH or cortisone on it as far as I know.

DR. JULIUS MCCALL (Cleveland, Ohio): A very interesting article was presented last year by Dr. Teilum, of Copenhagen, on some very excellent experimental work on mice, in which he fed these mice for a period of time on a heavy ascorbic acid diet. Then the spleens of some of the mice were opened; they did not show amyloid. By giving them cortisone, he was able to induce amyloid into the spleens of these mice very quickly.

Here is a part of his conclusions. He says that "Well-defined structural and functional phases in the natural diseases and in experimental disorders of the mesenchyma, which may in some way be associated with a disturbance of some enzyme system, such as ribonuclease, hyaluronidase, seem to be controlled by an interaction of a hormone of the adrenal cortex of cortisone type with ascorbic acid.

These findings offer an approach to the elucidation of the following: (1) the pathogenesis of amyloidosis, (2) certain apparently antagonistic effects of cortisone and ascorbic acid on the mesenchymal tissue, and (3) the mechanism of action of cortisone and ascorbic acid on mesenchymal tissue in general.

DR. H. W. SCHMIDT (Rochester, Minn.): I should like to thank the men who discussed this paper, because I think the subject is most interesting.

In answer to the question of whether or not primary systemic amyloidosis and tumor-forming amyloidosis can co-exist, I was very fortunate in having Dr. Starke sitting beside me, because he reviewed our cases of laryngeal amyloid deposits. He stated that one patient in the group whose cases he reviewed had primary systemic amyloidosis.

In all 3 cases that I reported the patients are still alive, so my colleagues and I are not sure whether they might have some evidence of amyloidosis in other places in the body or not.

In Case 3, we referred to a circumscribed nodule in the liver which the patient has had since 1948 without a change. We speculated as to whether or not this might be a deposit of amyloid, but in view of the fact that all 3 patients are living—and that with the use of all the tests that we have available, we cannot prove that primary systemic amyloidosis is present— we have to assume at this stage that they have localized deposits of amyloid. The blood of all 3 shows normal values for proteins, and normal A-G ratios.

I certainly agree with Dr. Holinger that the most conservative type of treatment should be utilized.

In Cases 1 and 2 the masses could not be proved to be amyloid, bronchoscopically, because we could not get specimens for biopsy. In Case 3 we did prove it, bronchoscopically; our chief problem in this case was the obstruction of both of the patient's major bronchi, and a decreasing airway.

Clinical Notes

LXXX

THE ASSOCIATION OF CONGENITAL CHOANAL ATRESIA AND CONGENITAL HEART DISEASE

REPORT OF TWO CASES

FRANCIS H. MCGOVERN, M.D.

DANVILLE, VA.

Inasmuch as a survey of the American literature did not disclose a record of the association of congenital choanal atresia and congenital heart disease, a report of two such cases was deemed of interest. In my paper[1] on some of the physiological and hereditary aspects of choanal atresia, mention was made of the abnormalities reported by others as being associated with congenital occlusion of the posterior nares. The abnormalities included high arching of the palate, asymmetry of the face, bifid uvula, congenital coloboma of the iris, double tragus, double congenital aural fistula, polydactylism, and flat nose with separation of the cartilages in the tip.

In the past three years I have seen five additional cases of congenital choanal atresia, two of which presented congenital heart lesions.

REPORT OF CASES

The first case was a premature, newborn infant. Consultation was requested because of cyanosis, weak cry and grasping respirations noted soon after birth. A diagnosis of complete bilateral bony atresia of the posterior nares was made. As an emergency measure, the atresia was corrected surgically, using the intranasal approach. The following day the cyanosis and feeding difficulty appeared to be improved, but in the succeeding days it became evident that the intermittant bouts of cyanosis were increasing in number and extent. A congenital heart lesion was suspected. X-ray examination of the chest showed an enlarged heart, almost filling the thoracic cavity. Despite continuous oxygen therapy and other supportive measures, the infant died twelve days after birth.

The postmortem examination showed congenital heart disease, a patent ductus arteriosis, hypertrophy of the heart, congenital atresia of the choana corrected surgically, and petechial hemorrhages of the lungs. Death was attributed to the patent ductus arteriosis.

The second patient was an eight-year-old girl. She complained of inability to breathe through the left side of her nose and constant secretion coming from the left nostril. Examination showed a complete bony atresia of the left posterior nares. The septum was deviated to the left; the nasal mucosa was pale and boggy and the left nasal fossa was filled with mucoid material. X-ray examination showed the sinuses to be fully developed; the left antrum, ethmoid and sphenoid were hazy. Lipiodol instilled into the left fossa demonstrated the occlusion radiographically. The general physical examination presented a well developed, well nourished intelligent girl. The tonsils and adenoids were moderately enlarged. There was no evidence of cyanosis, edema, or clubbing of the fingers. The lungs were clear. The heart was not enlarged to percussion; the rhythm was regular. There was a grade three systolic murmur, maximum at the second left interspace. The murmur was audible in the left axilla in the back. The first sound was rather loud at the apex. Flouroscopy showed the heart within normal limits of size and the contour was normal.

The cardiological consultant felt that this child undoubtedly had congenital heart disease and the specific lesion was probably a defect in the intraventricular septum.

The choanal atresia was corrected by the intranasal route.

COMMENT

It has been stated in the past that other congenital anomalies are occasionally associated with the choanal atresia. To this list is added congenital heart disease.

There was no evidence in these two cases of an hereditary factor. In this regard it is interesting to note that Dickson[2] has recently recorded bilateral choanal atresia in twin sisters. This report duplicates the family I reported in my previous paper, and adds to the evidence that this congenital anomaly has an hereditary tendency.

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LXXXI

CONGENITAL CYST OF THE BASE OF THE TONGUE

REPORT OF A CASE

JULIUS A. HOWELL, M.D.

AND

THOMAS C. PRINCE, M.D.

WINSTON-SALEM, N. C.

According to a recent article by Sharpiro,[1] only 12 cases of congenital cysts on the base of the tongue have been reported in the literature, and one of these was really a cyst of the body of the tongue. All of these cysts were found in infants or very young children. Shapiro postulates that the embryologic origin of such a cyst may be the thyroid, the lingual duct, the mucous glands, crypts of the lingual tonsil, fetal cell remnants, or a dermoid.

While this condition is rare, its recognition is important, since the measures necessary to correct it are simple and may be life-saving. These cysts, by pressing on the larynx, may cause respiratory obstruction and death. In eight of the 12 reported cases the diagnosis was made at autopsy. This fact emphasizes the necessity for being aware of the possibility of this condition in all cases of respiratory distress in infants.

Symptoms, which may begin in earliest infancy, vary in severity from time to time. A peculiar cry, inspiratory stridor, and intermittent attacks of complete respiratory obstruction with cyanosis may be noted, and the latter attacks sometime prove fatal. Feeding problems, including regurgitation, dysphagia and inability to suck are frequent and are accompanied by weight loss or failure to gain at a normal rate. Partial relief from the above symptoms may be afforded by proper positioning of the patient.

A cyst of the base of the tongue can usually be visualized by depressing the tongue with a tongue blade, or palpated by inserting the finger into the back of the mouth. If necessary, the diagnosis

From the Department of Otolaryngology, Bowman Gray School of Medicine of Wake Forest College and the North Carolina Baptist Hospital, Winston-Salem, N. C.

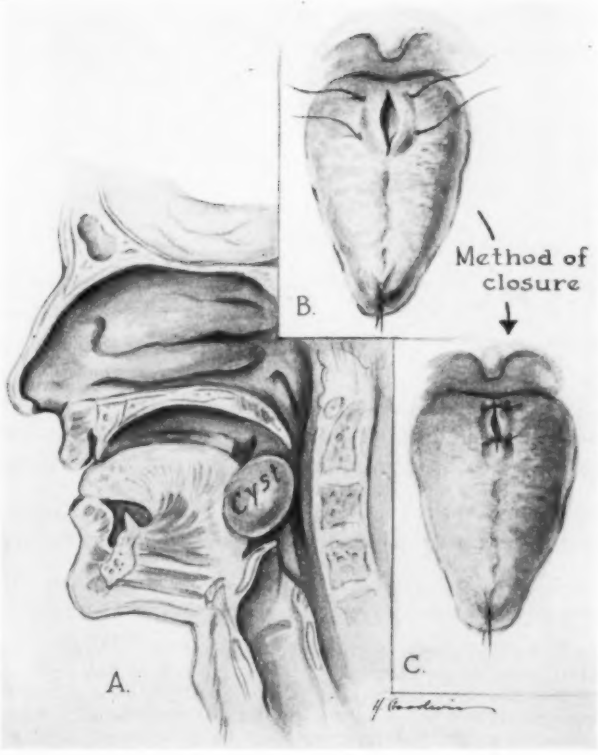

Fig. 1.—A. Hemi-section of nasopharynx indicating location of cyst at base of tongue. B. Dome of cyst has been excised with removal of an elliptical portion of the cyst wall. Sutures placed through base of tongue at anterior and posterior aspects of cyst. C. Sutures have been tied, obtaining hemostasis and leaving area for drainage between sutures.

Fig. 2.—(42 x) Microscopic section of tissue removed from dome of cyst. Arrows indicate squamous epithelium which comprised the lining of the cyst.

may be confirmed by direct laryngoscopy. Shapiro points out the importance of differentiating between a cyst and a lingual thyroid.

Treatment consists of simple incision and drainage of the cyst. In the majority of cases this results in complete disappearance of the mass.

CASE REPORT

A 15-week-old female infant was first examined on the pediatric service at the North Carolina Baptist Hospital. She was said to have had rapid respiration and minimal stridor since birth. The respiratory sounds were described by the mother as "crowing" in type. At the age of three weeks a cold and cough developed. Coughing sometimes made the child become cyanotic, and during the week prior to admission the paroxysms of coughing and episodes of cyanosis became more frequent. Since birth the child had refused on numerous occasions to swallow her feedings, in spite of frequent formula changes.

The child was born at seven and one half months of gestation, and her birth weight was 5 pounds, 3 ounces. Development had been slow, supposedly because of repeated "colds" for which she had received 21 injections of penicillin.

Physical examination revealed a poorly nourished and poorly developed white female infant appearing both alert, chronically ill and irritable. The temperature was 99 degrees rectally, pulse 120, respiration 38 per minute. The child weighed 8 pounds, 3 ounces and measured 22 inches in length. The remainder of the physical examination was essentially negative except for a cystic mass on the base of the tongue measuring 2 x 3 cm which could be partially seen and easily palpated.

Under endotracheal ether anesthesia the dome of the cyst was excised and an elliptical segment of tissue measuring approximately

1 cm x 3 mm was removed. No. 000 plain catgut sutures were placed through the base of the tongue at the anterior and posterior aspects of the cyst and were tied, leaving space for drainage between the cut edges of the dome. The operative area was slightly edematous for two days after the procedure but there was no recurrence of the cyst. After the edema subsided, the patient began taking feedings well without regurgitation. She had no further respiratory difficulty, and was discharged on the sixth postoperative day. One month after discharge she weighed 11 pounds and 12 ounces. Three and one half months after discharge from the hospital palpation revealed no recurrence of the cyst, and the mother reported that the child was having no difficulties with breathing or swallowing.

SUMMARY

Congenital cysts on the base of the tongue, while rare, should be considered as a possible cause of respiratory difficulties or feeding problems in infants. Correction of the condition is simple and may be life-saving.

The condition can usually be diagnosed by visual examination with the aid of a tongue depressor, or by palpation of the cyst with the finger. Direct laryngoscopy may be used to confirm the diagnosis.

Complete disappearance of the cyst usually follows incision and drainage or removal of an elliptical portion of the dome of the cyst.

The case of a 15-week-old infant who was found to have a cyst on the base of the tongue is presented and the operative treatment and results are described.

NORTH CAROLINA BAPTIST HOSPITALS, INC.

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LXXXII

A LENS AS AN AID IN JUDGING THE PROGNOSIS IN NEOPLASTIC SURGERY

ALBERT P. SELTZER, M.D.

PHILADELPHIA, PA.

The nose, because of its prominence in the human profile, is the most important feature of the face. It can make or mar the first impression of a person, and in the event of mutilating injury, or with congenital malformations, or exaggerated departures from the usual in size and shape, the surgeon must be assured that any operation for reconstruction of the nose will result in improvement of contours. Consequently all the means at his disposal should be employed to strengthen the assurance that the operation will prove successful.

In examining the nose before undertaking cosmetic surgery, the characteristics of the patient's skin are of first importance in judging the outlook for rapid and satisfactory healing. These can be determined roughly by naked eye inspection, together with manual manipulation.

By careful manipulation, the sense of touch reveals the variation in skin thickness, in its elasticity, the abnormal presence of dermal fibrous tissue, and the presence of minute intradermal nodules.

This method of examination also shows that there is a distinct difference in the character of the normal skin over the bridge of the nose from that overlying the lower, cartilaginous portion. The former is relatively thin, more elastic and movable; when stretched, it retracts promptly to its original position. In contrast with this, the skin over the lower, movable part of the nose is less elastic and cannot be easily lifted from the original position; it is firmly bound to the underlying tissue. This peculiarity is easily demonstrated over the lower two thirds of the nasal dorsum. The skin over the glabellar region is also thicker than that over the bridge.

A clearer understanding of these differences between the skin over the upper third of the nose and that over the lower two thirds

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From the Department of Plastic and Reconstructive Surgery, St. Luke's and Children's Medical Center.

Fig. 1.

can be clarified, by a brief review of the embryology of the nose, and the histology of the nasal skin.

It will be sufficent to remember that the nose of the embryo develops from different centers of growth of the primitive face region. The upper part of the nose if formed by the downward extension of the fronto-nasal process, while the lower part of the nose is derived from the maxillary processes.

When the maxillary processes reach the point of uniting near the midline, with the fronto-nasal portion, as soon as the fusion of these parts takes place, the mesenchyme (embryonic fibrous tissue) of the former begins to infiltrate the fronto-nasal portion, and thus changes its original nature. This type of tissue then forms the lower nose, over the cartilages, and completes the oral opening by also forming the upper lip. This same type of tissue is reflected within the lower nasal openings to form the vestibule. Thus the lining of this part of the nares is similar to that of the lip, in contradistinction to that above the *limen vestibuli*, which is of mucosal character.

These peculiarities of development of the nose afford an explanation for the mobility of the skin over the upper part of the nose, and the difference from that over the lower segment, where it is considerably thicker and closely adherent.

To complete the examination of the skin over the nose, the writer has recently found that going over the field with a jeweler's lens is a notable aid in determining the finer skin changes that are not visible on simple inspection, and so are not usually considered.

A lens with a 3x magnification and focal distance of 3½ inches, or a 5x lens with two inch focal distance, is suitable, though the higher magnification gives better detail. One can clearly see the size and relative number of the pores; also, whether the secretions are impacted, or being normally discharged, which is not evident without the use of the lens. Various other minute defects of the skin can also be detected by this means, as foci of early epidermal hyperplasia, or of capillary overgrowth; conditions that may all be of considerable significance in skin surgery. The degree of the changes of aging skin can also be more accurately determined under magnification.

A considerable experience with these methods of examination of the skin has demonstrated that when the tissues are soft and pliable the prognosis is better than when they are tickened and more rigid on manipulation, and firmer to the touch. These conditions are more common with age, although the skin may age more rapidly in some persons than in others. Thus the calendar age is of less significance than the physiological age of the skin itself. Such changes may be seen soon after thirty years of age, or they may be delayed until considerably later. The maintenance of the capillary blood supply of the skin is an important factor in determining the degree of change.

The prognosis in the presence of numerous pores, particularly if they are enlarged and show increased secretion, is much less favorable than where the opposite is true. Where there is the suggestion of the development of acne, when the skin is examined with the lens, the outlook for successful surgery is considerably diminished.

Years of personal experience in surgery on the nose have indicated to the writer that the most careful pre-operative examination of the skin, by all the means available, is a definite help in prognosticating the outlook for successful results. For this reason the use of the lens is suggested to others whose interests lie in this field, and who may also find it an aid in preparing for operations on the skin.

2104 Spruce St.

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Abstracts of Current Articles

EAR

Glomus Jugulare Tumor of the Middle Ear.

Brown, Lester A.: The Laryngoscope 63:281-292 (Apr.) 1953.

The symptoms, clinical findings and treatment of six proven cases of glomus jugulare tumor of the middle ear observed for periods from three months to four and one-half years are presented. Five additional patients with similar symptoms and findings but without confirmatory biopsies are discussed. The earliest and most constant symptoms were tinnitus and impaired hearing. Other presenting complaints included hemorrhage, discharge, facial weakness, vertigo, headache and itching deep in the ear canal.

The most constant diagnostic finding was a "pulsation sign" which could be elicited in almost every case. Invariably, there was a visible mass in the ear. In some cases, it appeared to be incorporated into the tympanic membrane, presenting a deep red, or blue mulberry-like roughness. In other cases, the impression was given of an aural polyp adherent to or protruding through the drum membrane. Hearing varied from slight to total impairment. Facial nerve weakness was present frequently. In the early stages of the disease no radiologic changes were evident, but as the disease progressed, some clouding of the mastoid cells occurred and late in the disease there was decalcification of the trabeculae and coalescence.

Treatment consists in surgical enucleation of the tumor mass followed by x-ray therapy. The post-auricular incision appeared to offer some advantages, e.g., wider exposure and avoidance of early bleeding from the mass. Since the tumor was located in the middle ear, this part of the procedure was left to the last. A transfusion of blood was given duirng the course of the operation because of profuse bleeding. X-ray therapy was started 7-10 days following operation with a dosage varying from 2500 to 4800 R.

SENTURIA.

Hearing Impairment in Children.

Kinney, Charles E.: The Laryngoscope 63:220-226 (Mar.) 1953.

An attempt is made to analyze and interpret the otologic and audiologic findings of 3,622 deafened children screened from the

city school system and observed over a period of fifteen years. Ages ranged from 2½ to 11 years with a mode between 8 to 11 years.

Ten percent of the children were classified as having no usable hearing in either ear while 17 percent were found to have normal hearing. This left 2,628 cases to be studied. These were divided into the following categories: conduction loss, unilateral total deafness, and partial perceptive loss in one or both ears. In those instances where mixed deafness was present, the case was classified in only one category according to the predominating type of deafness.

In the breakdown of the conductive deafness group, it is interesting that 18 percent showed active otitis media, 51 percent showed healed middle ear disease, 23.5 percent were classified as "tubal and or adenoid" while an additional 2 percent showed middle ear effusions. It would have been interesting to correlate the nasopharyngeal findings in this 94 percent of the cases.

The author attempted to established the cause of the unilateral total perceptive deafness. The high incidence of "probable meningitis" and "possible measles" is interesting. As would be expected it was not possible to determine the cause of partial perceptive deafness in almost half of he patients. The remaining categories include the usual groupings, with unusual emphasis on measles.

SENTURIA.

Facts and Fallacies of Bone Conduction.

Weille, Francis L., and Gargano, Samuel (by invitation): *The Laryngoscope* 63: 182-211 (Mar.) 1953.

The authors attempt to translate the technical scientific jargon of the research physicist, pyschologist and mathematician into understandable medical and otologic concepts. Much of the information presented is transcribed from the excellent works of Békésy and Bárány. Audiologic observations are reported on four patients who had essentially normal hearing on one side and total unilateral surgical destruction of the function of the membranous labyrinth in the contralateral ear.

It is the impression of the authors that much can be learned from the intelligent use of bone conduction audiograms, but that careful use of quantitative masking is required. At the present time there is no accepted bone conduction standard and substantial variations may occur from one instrument to another.

SENTURIA.

Study of Fifteen Cases of Agenesis of the External Auditory Canal. Techniques Employed and Suggested.

Tato, J. M., Bello, J. A., and Castellano, J. L.: Acta Oto-Rino-Laryngol. Ibero-Amer. 4:85, 1953.

Although early treatment is not essential in cases of unilateral developmental defects, bilateral agenesis should be corrected surgically before the age of three or four years, in order to improve hearing and thus ensure normal intelligence, speech and educational capacity, and also to avoid complications, especially meningoencephalitis, resulting from otitis or mastoiditis in the deformed ear. Esthetic correction of the auricle should be carried out later, for psychologic reasons. There is no parallelism between the degree of atresia or malformation of the auricle and that of the auditory canal, or antrum. Roentgenographic examination prior to surgery is essential to determine the degree of malformation. Absence of the mastoid antrum and of the middle ear is no contraindication to surgery.

Otologic correction consists first in trepanation of the mastoid bone, which is simple if the pneumatic cells are well-formed, and the antrum large, so that access to the cavity of the middle ear is easy, but difficult if the process is sclerotic and the antrum underdeveloped or absent. If pneumatization is good, the cavity is freed of irregularities, to prepare the surface for the subsequent skin graft. If there is sclerosis, the entire dura mater is exposed and raised with an elevator, to afford access into the antrum. If the dura mater lies deep, fenestration of the posterior semicircular canal is carried out, or a free bone graft taken from the temporal bone is inserted below the dura mater to raise it, followed by atticomastoidotomy, and the denuded dura mater is covered with a free skin graft.

Fenestration is the second stage, but is not necessary if the stapes is free and the round window is open. Extirpation of the incus is in itself sufficient to ensure functional improvement.

Thirdly, a free skin graft shaped like the finger of a glove is inserted.

Plastic correction may be by no means of a skin graft attached to the retroauricular region by a pedicle, and forming a tube, which is introduced into the bony cavity. The disadavantage is that the skin does not cover the entire raw bone surface, and that granulation tissue forms on the exposed areas. Furthermore, the later detachment of the pedicle leaves scars in the skin of the mastoid area which is to be used for the reconstruction of the auricle. The authors therefore use a free skin graft 0.5 mm in thickness (thus made up of dermal

and epidermal layers) obtained by means of a dermatome or bistoury from a non-hairy area on the thigh, shoulder, or, preferably the inner surface of the arm, which is free from sebaceous glands. It is advisable not to remove the entire thickness of the skin in the donor area, to avoid large scars, and also to prevent the development of hair or of sebaceous secretion in the graft.

A rectangular piece of skin is formed into a cylinder or cone, according to the size of the bony cavity to be covered. It is atached by an interrupted suture, with the knots toward the lumen of the passage. It is closed at one end, like the finger of a glove, with the bleeding surface outward, and is fitted to the cavity and packed with small pieces of sponge rubber or cicatul, under pressure. The margin of the cylinder is sutured to the outer margin of the orifice.

One must guard against cicatricial stricture of the newly-formed meatus, which may necessitate dilatation by means of a funnel-shaped acrylic prosthesis. The orifice should be large enough to allow for this cicatricial narrowing of the skin ring lining the passage.

HIGBEE.

A Study of the Visual Perception of Deaf Children.

Mykleburt, H. R., and Pnutten, M.: Acta-Otolaryng. Suppl. 105.

This is an exhaustive study aimed at determining whether deaf children differ from normal hearing children in certain aspects of visual perception. Educators of the deaf have concentrated their efforts in applying remedial techniques to overcome receptive and expressive language defects, although it has been shown that deaf children are handicapped in other areas such as reasoning, vocal competence and emotional stability. Such children have been found to have a high incidence of subnormal vision and of disturbed motor control. This indicates the importance of careful individualization to the end that defects other than that of impaired hearing be given consideration.

The author's studies show that deaf children may experience difficulties in perception such as in learning to read. They conclude that "deafness causes an alteration in the normal response modes of the organism, and that this alteration includes certain arresting disruptions in visual perceptual organization." Their study "offers further evidence of the interrelatedness of functions within the unitary organisms." Perception reflects the psychoneural state as a whole and not merely the excitation of the sensory end-organs.

HILL.

Vestibular Symptoms in Carbon Monoxide Poisoning after Unilateral Ligation of the Common Carotid Artery.

Floberg, Lars-Erik: Acta Oto-Laryngologica Suppl. 106.

The author, by means of well planned experimental projects, has answered several questions regarding the effect of carbon monoxide on vestibular function. It is shown that acute or chronic CO poisoning have no effect on the vestibule of the intact animal (cat). CO exposure following ligation of one carotid artery does result in vestibular damage. Studies of similar nature following destruction of the labyrinth directly or by streptomycin yielded results identical to those seen before destruction of the labyrinth, demonstrating the location of action to be central to the vestibule and the vestibular nuclei. To complete the study, the author demonstrates clearly that these experiments yield the same results if hypoxia is substituted for carbon monoxide in each instance.

The project is well organized and excellently presented.

GOODOF.

NOSE

Bilateral, Congenital Bony Choanal Occlusion.

Ferreras, E.: Acta Oto-Rino-Laringol. Ibero-Amer. 4:45, 1953.

Reports that choanal occlusion is relatively rare may be misleading; most infants born with complete occlusion of the posterior nares die at birth, due to asphyxia and inanition, and these neonatal deaths may be erroneously attributed to forceps or birth injuries. The incidence is slightly higher in females than in males.

The condition has been variously attributed to: (1) excessive proliferation of the otherwise normal hard palate and vomer; (2) persistence of vestigial embryonic buccanasal membrane or of the fibromuscular velum of the soft palate; (3) abnormal asymmetry of the two cavities of the posterior nares. These anomalies may be inherently developmental, or due to intrauterine disturbances.

Various types of occlusion have been described: fusion of the vertical plate of the palate with the vomer leaving only an infinitesimal opening, or none at all; exclusive proliferation of the horizontal portion of the palate, forming a wall which extends upward and obliterates the choana; abnormal growth of the nasal septum, which forms folds and blocks the nasal cavities. The choanae are not simple openings allowing communication between the nasal fossae

and the pharynx, but rather cylindric infundibular canals, and the majority of the occlusions occur at the point of junction of the two superimposed canals.

Obstructions due to malformation may be bony, membranous, cartilaginous, mixed, or calcareous. The bony obstruction is by far the most frequent. It may effect the base of the sphenoid bone, the pterygoid process, the palate or the vomer. The nasal septum is frequently deviated, and the palatal vault may be raised. In the case of a 38-year-old patient with bilateral occlusion, roentgenographic examination revealed maxillary atrophy. Histologically the occluding bone tissue does not differ from the normal.

Bilateral complete occlusion is usually fatal in the case of the newborn. If the occlusion is unilateral or incomplete, respiratory disturbances resembling those of laryngeal stridor result. Nursing is difficult, and therefore the first month is the most dangerous period. Older children develop rhinolalia clausa, mouth-breathing, restless sleep, noctural enuresis, an adenoidal expression, collapsed nostrils, and facial asymmetry. In the case of adults, the obstruction results in persistent mucus, complete or partial loss of the sense of smell, violent headaches, defective hearing, dryness of the larynx and pharynx, scintillating scotoma, facial asymmetry due to deformation of the maxilla and dental arch, and occasionally hypertension and menstrual disturbances.

In infancy and early childhood, the diagnosis based upon the abnormal respiration, especially during nursing, can be varified by exploration with fine probes, or by instillation of methylene blue or other stains. In the case of older children and adults, posterior rhinoscopy, contrast roentgenography, and transillumination are of help.

Surgical correction is usually successful and rarely dangerous, and restores the olfactory and auditory functions. Preceding surgery, it is essential to determine the exact location, extent, and nature of the obstruction, to avoid trauma and perforations. In addition to trocars and probes, galvanocautery is sometimes useful.

In the author's case (adult), the bony obstruction was removed by trepanation of the angle of the nasal septum and the nasal floor.

HIGBEE.

PHARYNX

Malignant Lesions of the Tonsil.

Parshall, Dale B., and Stenstrom, K. W.: Radiology 60:564-571 (Apr.) 1953.

One hundred and three cases of cancer of the tonsil are reviewed. These were treated by x-radiation during the period from 1926 through 1950. Supplemental radon implantation was used in the primary tumor and lymph node metastasis. Three and five year survivals compare favorably with other series reported. Histologic characteristics, type and amounts of total radiation doses used, age, incidence and survival rates are discussed. Four figures, six charts and fifteen references are included.

JORSTAD.

LARYNX

Cysts in the Ventricular Area of the Larynx.

Laff, Herman I.: The Laryngoscope 63:227-240 (Mar.) 1953.

The author presents a survey of cystic swellings in the ventricular area of the larynx. It is postulated that the congenital variety as found in infants is most likely due to a developmental error arising in the ventricle or saccule. Some such cysts found in adults are said to have a similar origin while others probably begin as laryngoceles.

Three adults with ventricular cysts form the basis of this report. The usual symptoms consist of hoarseness or dyspnea, cough, a desire to clear the throat and a sense of fullness. The mirror examination reveals a smooth, shiny, round mass occupying the region of the ventricular band alone or involving the adjacent epiglottis, pyriform sinus, and aryepiglottic fold.

External extension of the cyst may cause difficulty in differentiation from carcinoma of the ventricle with metastases to the cervical glands. Early surgical interference is advised. Preliminary tracheotomy and evacuation of the contents of the cyst is considered advisable. Recourse to thyrotomy may be desirable to obtain total removal of the cyst.

SENTURIA.

Tracheotomy in Cancer of the Larynx. Latin-American Committee for the Study of Laryngeal Cancer.

Latin-American Committee For the Study of Laryngeal Cancer: Rev. Peruana Otorrinolaringol. Oftalmol. 2:33 (Jan.-June) 1952.

Tracheotomy is indicated in cancer of the larynx either as an early preventive measure, to avoid obstruction, or as an emergency operation during an acute suffocative crisis.

The classical procedure consists in making a vertical incision in the median line of the neck, separating the muscles to expose the cricoid cartilage, and the thyroid isthmus, and opening the first or second ring of the trachea to introduce a cannula. The method has the following disadvantages: (1) The incised superficial tissues may come in contact with tracheal secretions, resulting in suppuration and scarring. This can be avoided, if the tracheotomy is to be permanent, by suturing the skin of the incision to the opening made in the trachea; (2) when the cannula is withdrawn, the thyroid isthmus tends to revert to its original position, blocking the tracheal opening. To prevent this, the isthmus should be severed in the midline, and each side attached to the surface of the incision; (3) The tracheal cartilage may be damaged by the cannula and become necrotic, or the ring may contract, constricting the tracheal lumen. This can be avoided if, in place of section of the trachea, a disc approximately 11 to 12 mm in diameter is removed from the anterior wall, to admit the cannula. The tracheotomy incision may also be horizontal or curved.

It is advisable to give oxygen and carbogen prior to surgery, and to introduce a 4-mm tracheotomy tube, under local anesthesia.

In acute cases, if there is danger of asphyxiation, the patient should be in a sitting position, adequately supported, rather than recumbent, to facilitate breathing.

Surgery can be carried out under novocain anesthesia. Care must be observed to avoid pressure on the trachea and larynx, so as not to produce reflex spasms.

Before puncture of the anterior wall of the trachea, it is advisable to inject 1 cc of a five percent solution of cocaine into the trachea, to forestall coughing. Following tracheotomy a respiratory stimulant, such as lobeline, is given.

In an acute emergency, the patient is placed on his right side, a large incision is made over the lower thyroid border, the surgeon introduces the index finger of his left hand, and lifts the lower border

of the cricoid cartilage. The bistoury is slipped under the finger, blade downward, and an incision four cm long is made downward in the middle line of the trachea. The cannula is introduced, hemostasis assured, and the thyroid isthmus is sutured.

HIGBEE.

MISCELLANEOUS

A Case of Malignant Tumor of the Maxilla. A Study of Osteogenic Sarcomas.

De Villasante, J. Portella: Acta Oto-Rino-Laringol. Ibero-Amer. 4:5, 1953.

The case cited, of osteogenic sarcoma of the upper jaw, differs from the classical tumors of this nature, which usually have a predilection for the diaphyses of the long bones of the extremities.

The patient, a 12-year-old girl, had suffered a blow on the nose at the age of six years, which had resulted in temporary pain and swelling, but no bleeding. Four years later a slight, painless swelling of the right cheek had appeared, gradually increasing in size over the ensuing two years, and resulting in asymmetry of the right malar region, outward and upward deviation of the right eye-ball, enlargement of the right nostril and right buccal commissure. Further complications were sharp, lancinating head-ache, no pain on mastication but vomiting, chills, dyhphagia, and loss of weight. There was no history of catarrhal rhinitis or inflammation, and no pathologic secretion.

Palpation revealed a well-defined, smooth, bony growth in the right malar region, without adhesions to overlying tissues, and painless on pressure. The right nasal fossa was constricted, the turbinate congested and in contact with the septum, so that cocaine-gauze could not be introduced. Exploration was not possible without pain and bleeding, and breathing was impossible on that side.

Transillumination and roentgenography of the right maxillary sinus showed complete opacity extending to the lower rim of the orbit, the zygomatic arch, external wall of the right nasal fossa, and in an irregular line to the upper dental arch. On introduction of a trocar, the sinus was found to be completely filled with a partly spongy tissue, which yielded a small amount of blood on aspiration.

There were no other physical or serologic abnormalities.

Surgical intervention was carried out by the nasal transmaxillary technique of Moure. The external paranasal incision was carried

as far as the ascending apophysis of the upper jaw and the pterygomaxillary region and the right infraorbital border, combined with a sublabial incision (Caldwell-Luc). The spongy bone tissue was readily and completely removed with a curette, together with a part of the jaw-bone, right nasal bone, and the ascending process of the maxilla. Following electrocoagulation of the entire operative field, the cavity was cleansed with solution under pressure, and the incision was closed, allowing for drainage into the nasal fossa. Postoperative treatment consisted in massive doses of penicillin, and deep radiotherapy.

Roentgenographic examination 14 days and again 35 days later showed complete recovery.

The excised tumor had the histologic characteristics of an osteogenic fibrosarcoma.

In the absence of the suppurative sinusitis and purulent nasal discharge usually associated with malignant tumors of the maxillary sinus, chronic sinusitis could be excluded as an etiologic factor. The sarcoma was attributed to the previous trauma during the period of bone growth.

The prognosis of traumatic sarcomas depends upon the age of the patient, the duration of the condition, its location (type of bone affected), operative technique, and degree of malignancy. Individuals younger than 20 years have poor resistance to malignant tumors.

The author considers biopsy of questionable value as a diagnostic aid, due to the danger of accelerating malignant cell proliferation, or of causing severe hemorrhage. He recommends irradiation in conjunction with surgical intervention, especially in cases of fibrosarcoma. Chondrosarcomas are refractory to all forms of treatment. The danger of metastases following surgical intervention is greatly decreased by electrocoagulation of the operative field, and by preoperative and postoperative use of radium.

HIGBEE.

Postoperative Thrombophilia.

Warren, R.: N. E. Jour. Med. 249:97-107 (July 16) 1953.

This is an excellently done study on the causes of postoperative thromboembolism pertinent to all fields of surgery. The mechanism of coagulation of blood, with the factors involved, is discussed, together with the part played by anaesthesia an surgical trauma. During

the first week of the postoperative period a thrombocytopenia occurs while the coagulation time is shortened. During the second week thrombocytosis occurs and post-thrombin consumption and anti-thrombosis return to normal. It is felt that the factors of stress and tissue trauma are most significant that those of anesthesia and hemorrhage.

HILL.

Books Received

Treatment of Respiratory Emergencies Including Bulbar Poliomyelitis.

By Thomas C. Galloway, M.D., Professor Emeritus of Otolaryngology Northwestern University Medical School, Attending Otolaryngologist Evanston Hospital, Evanston, Ill., Formerly Attending Otolaryngologist Cook County Hospital, Chicago, Ill. Pp. 94, Crown 8 vo., Charles C. Thomas, Springfield, Ill., 1953.

Medical men in general and more especially the members of the laryngological societies who, through the years, have received with absorbing interest Dr. Galloway's contributions on the different aspects of anoxia, will rejoice at the appearance of this informative little monograph.

The book is packed with useful, often vital, information and the exposition is clear and simple—as is usual with this author.

Meniere's Disease.

By Henry L. Williams, M.D., Consultant in Section on Otolaryngology and Rhinology, Mayo Clinic. 8 vo., Pp. 349, Charles C. Thomas, Springfield, Ill., 1952. (Price $7.00)

A very complete compilation of the literature of Ménière's disease, presented with a discrimination which only a critic of this author's experience could achieve.

The first chapter, dealing with a review of Ménière's original papers and the misconceptions still current regarding them, is alone worth the price of the book. Other chapters deal adequately with anatomy, pathology, diagnosis and treatment, and the fundamental identity of allergy and autonomic dysfunction. Copious references.

The index is sketchy, which is a pity since this is essentially a reference work.

Advances in Oto-Rhino-Laryngology.

Edited by L. Rüedi, M.D. Pp. 263, illustrated, S. Karger, Ltd., Basle, Switzerland, 1953. (Price 36 Swiss fracs)

This first volume of a series, is devoted in its entirety to the vestibular apparatus.

It is in three parts: I. Methods of investigating the balancing organ, by L. B. W. Jongkees, Amsterdam; II. the Standardization of

methods, by M. Arslan, Padua; III. Physiopathologic problems, by E. Wodak, Tel-Aviv.

The presentation is in text-book style; in German.

Certain New Ideas On Vertigo (Quelques Notions Nouvelles Sur Le Vertige).

By Rene Mayoux, M.D., Professor agregé a la Faculteé de Medicine de Lyon. Pp. 140, 8 vo., Masson et Cie, Paris, 1953. (Price 865 fr.)

The Conception of Disease, Its History, Its Versions and Its Nature.

By Walther Riese, M.D. 8 vo., Pp. 120, Philosophical Library, New York, 1953. (Price $3.75)

The Pharynx: Basic Aspects and Clinical Problems.

Edited by Abraham R. Hollender, M.D. F.A.C.S., Professor of Otolaryngology, Emeritus, University of Illinois College of Medicine; Attending Otolaryngologist and Chairman of the Service Mount Sinai Hospital of Greater Miami; Consulting Otolaryngologist, Variety Children's Hospital, Miami, and St. Francis Hospital, Miami Beach, Fla. Pp. 560, 8 vo., illustrated, The Year Book Publishers, Inc., Chicago, 1953.

Diseases of the Esophagus.

By Philip Thorek, M.D., F.A.C.S., F.I.C.S., Associate Clinical Professor of Surgery University of Illinois College of Medicine; Diplomate American Board of Surgery; Professor of Esophageal Diseases and Clinical Surgery, Cook County Graduate School of Medicine; Member American Association of Anatomists; Fellow American College of Chest Physicians; Co-Surgeon in Chief American Hospital; Attending Surgeon Cook County Hospital; Senior Attending Surgeon Alexian Brothers' Hospital; Author of Text, "Anatomy in Surgery." Pp. 140, illustrated, J. B. Lippincott Company, Philadelphia, 1952. (Price $10.00)

Health Saboteurs.

By Robert William Davis, M.D. 8 vo., Pp. 306, illustrated, Pageant Press, New York, 1953. (Price $4.00)

Three hundred pages, leading up to this author's thesis, which is, in his own words, " . . . to advance the time for universal tonsillectomy to two years of age, or younger if indicated . . . There is no object in carrying a time bomb around any longer than necessary. In my own practice I usually advise operation before the child reaches his second winter—even though he is little over a year old—in order to lessen the risk of serious infections . . . " He fully agrees with Professor Brown, of Toronto, that tonsils may be removed, if

indicated "any time after the navel cord is tied" and predicts that in the future the tendency will be to operate much earlier than at the present time.

The Book of Health.

Compiled and Edited by Randolph Lee Clark, Jr., B.S., M.D., M.Sc., and Russell W. Cumley, B.A., M.A., Ph.D. Pp. 770, illustrated, Elsevier Press, Houston and New York.

A great big pretty-picture book, full of the most intimate details of medicine, surgery, obstetrics and pathology for the edification of the medically curious layman (and, unfortunately, the incipient hypochondriac).

Notices

ACADEMY HOME STUDY COURSES

The Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of ten months. Registrations must be completed before August 15. Detailed information and application forms may be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minnesota.

AMERICAN LARYNGOLOGICAL ASSOCIATION

CASSELBERRY PRIZE

A sufficient sum having accrued from the Casselberry Fund for encouraging advancement in the art and science of Laryngology and Rhinology, this sum is now available, in part or as a whole, for a prize award. Theses must be in the hands of the Secretary, Dr. Harry P. Schenck, 326 South 19th Street, Philadelphia, Pa., before March 1, 1954.

UNIVERSITY OF PENNSYLVANIA

Announcement is made of the personal fundamental course in Bronchology, Esophagology, Gastroscopy and Laryngeal Surgery at the Graduate School of Medicine, University of Pennsylvania. The course begins September eighth and continues through September nineteenth.

Courses are scheduled usually in January, June and September avoiding conflict with the meeting dates of the National Medical Societies.

Application may be made to Dr. Aims C. McGuinness, Dean, or to Dr. Gabriel Tucker, Chairman of the Department of Bronchology, Esophagology, and Laryngeal Surgery.

McGILL UNIVERSITY TEACHING HOSPITALS

A three-year training in Otolaryngology is offered which is designed to qualify the trainee for the examinations of Certification or Fellowship of the Royal College of Surgeons of Canada and for the examinations of the American Board of Otolaryngology, all three of which demand a minimum of three years special training.

The course consists of a rotation through three teaching hospitals of McGill University, viz. the Royal Victoria, the Montreal General and the Children's Memorial Hospitals. Included in the course is a four months full time period in General Pathology with special emphasis on the Pathology of the Ears, Nose and Throat.

For further information apply to the Chairman of the Department of Otolaryngology, McGill University, Montreal.

PAN-AMERICAN CONGRESS

Fourth Pan-American Congress of Oto-rhino-laryngology and Broncho-esophagology, Mexico City, January 1954. Dr. Ricardo Tapia Acuna, President, Dr. Chevalier L. Jackson, Executive Secretary, 1901 Walnut St., Philadelphia 3, Pa.

FRENCH CONGRESS OF OTO-RHINO-LARYNGOLOGY

The sixty-first French Congress of Oto-rhino-laryngology will be held in the Grand Amphitheatre of the Faculty of Medicine of Paris, October 26-29, 1953.

For complete information, please write Dr. H. Flurin, Secy., Cauterets (Hautes Pyrénées) or Dr. H. Guillon, Assoc. Secy., 6 Ave. McMahon, Paris 17, France.

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HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION OF THE AMERICAN MEDICAL ASSOCIATION

(As of September, 1953)

Audicon Models 400, 415, 530 and 615
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Audivox Models Super 67 and 70
Aurex Models L and M

Beltone Mono-Pac Model "Lyric"
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Dahlberg Model D-4 (Tru-Sonic)

Fortiphone Models 19LR, 20A, 21C and 22

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Goldentone Models 25, 69 and 97

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Maico Top Secret Model L
Moico UE Atomeer
Mears (Crystal and Magnetic) Aurophone Model 200
Micronic Model 303
Micronic Model Mercury
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Microtone Models T-10, T-612 and 45

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Otarion Model H-1, Custom "5"

Paravox Model D (Top-Twin-Tone)
Paravox Model J (Tiny-Myte)
Paravox Model Y (YM, YC and YC-7) (Veri-Small)

Radioear All-Magnetic Model 55
Radioear Permo-Magnetic (Multi-power)
Radioear Model 62 Starlet
Radioear Model 72
Radioear Model 82 (Zephyr)

Silvertone Models J-92 and P-15
Solo-Pak Model 99
Sonotone Models 900, 910, 920, 925, 940 and 966
Super-Fonic Hearing Aid

Televox Model E
Telex Models 97, 99, 200, 300-B, 400, 500, 952, 953 and 1700
Tonamic Model 50
Tonemaster Cameo Model

Unex Midget Models 95 and 110
Unex Models 200 and 230

Vacolite Models J and J-2

Zenith Miniature 75
Zenith Model Royal
Zenith Model Super-Royal
Zenith "Regent"

(All the accepted hearing devices have vacuum tubes.)

Accepted hearing aids more than five years old have been omitted from this list for brevity.

SEMI-PORTABLE HEARING AIDS

Ambco Hearing Amplifier (table model)

Aurex Semi-Portable

Precision Table Hearing Aid

Sonotone Professional Table Set Model 50

OFFICERS

OF THE

NATIONAL OTOLARYNGOLOGICAL SOCIETIES

American Academy of Ophthalmology and Otolaryngology

President: Dr. Derrick Vail, 700 N. Michigan Blvd., Chicago 11, Ill.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.

American Board of Otolaryngology

President: Frederick T. Hill, Professional Bldg., Waterville, Maine.
Secretary: Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.

American Broncho-Esophagological Association

President: Dr. Edwin N. Broyles, 1100 N. Charles Street, Baltimore, Md.
Secretary: Dr. F. Johnson Putney, 255 S. 17th St., Philadelphia 3, Pa.
Meeting: Statler Hotel, Boston, May 25th and 26th, 1954.

American Laryngological Association

President: Dr. Gordon F. Harkness, 2410 River Drive, Davenport, Iowa.
Secretary: Dr. Harry P. Schenck, 326 South 19th St., Philadelphia, Pa.
Meeting: Boston, Mass., May 20 and 21, 1954.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Francis LeJeune, Prytania and Olive Sts., New Orleans, La.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Meeting: New Orleans, La., April 28-30, 1953.

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Carl H. McCaskey, 20 N. Meridian St., Indianapolis, Ind.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3, Tenn.

American Otological Society

President: Dr. Frederick T. Hill, Professional Bldg., Waterville, Maine.
Secretary-Treasurer: Dr. John R. Lindsay, 950 East 59th St., Chicago 37, Ill.
Meeting: Boston, Mass., May 16 and 17, 1954.

The American Society of Ophthalmologic and Otolaryngologic Allergy

President: Kenneth L. Craft, M.D., 23 East Ohio Street, Indianapolis, Indiana.
President-Elect: Albert D. Ruedemann, M.D., 1633 David Whitney Building, Detroit 26, Michigan.
Secretary-Treasurer: Michael H. Barone, M.D., 468 Delaware Avenue, Buffalo 2, New York.
Meeting: Chicago, Illinois on October 16, 1953.

International Bronchoesophagological Society

President: Dr. Fernand Eeman, Ghent, Belgium.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U.S.A.
Meeting: Second International Congress of Bronchoesophagology, June 15th and 16th, 1953, Albert Plage, Le Zoute, Belgium.

Pacific Coast Oto-Ophthalmological Society

President: Leland G. Hunnicutt, M.D., 98 N. Madison Ave., Pasadena 1, Calif.
Secretary-Treasurer: John F. Tolan, M.D., 3419 47th Ave., N.E., Seattle 5, Wash.
Meeting: Honolulu, 1954.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Justo M. Alonso, Montevideo.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U.S.A.
Meeting: Fourth Congress of Oto-rhino-laryngology and Broncho-esophagology.
President: Dr. Ricardo Tapia Acuña, Mexico City.
General Secretary: Dr. Maximo Garcia Castaneda.
Time and Place: February 28th to March 4th, 1954, Mexico City.